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GAS TENSIONS IN THE TISSUES.

By ERIC W. SIBREE.

(From the Department of Medicine, University of Sydney.[1])

IN this and subsequent papers it is proposed to publish observations on the tensions of carbon dioxide and oxygen in the tissues of man and animals. The "gas depot" method has been used, the general lines of the investigations of J. Argyll Campbell being followed. This investigator has published a great amount of work on animals, chiefly rabbits, and it is my object to apply these studies to the human subject. The gas tensions of the tissues will be correlated with those of the respiratory system in physiological and pathological conditions. The present investigations on animals have been carried out as a guide to human studies and as a check on previously published findings. The rather fragmentary observations on gas tensions in the human subject already made will be discussed in their appropriate places.

Most of the investigations by Campbell on gas tensions in the tissues have been reviewed in *Physiological Reviews*.[5] Campbell gives an historical survey of the subject and a general discussion of methods of investigation together with his own findings in animals.

The anatomy and physiology of the factors concerned in the exchange of gases between the atmosphere, alveolar air and arterial and venous blood have been studied widely. As pointed out by Campbell, however, comparatively little interest has been taken in the relation of the tissues to the respiratory system. Although these relations are

[1] Work done with the aid of a grant from the Australian National Health and Medical Research Council.

fairly simple, it is felt that some points require further emphasis than they have yet received to complete the understanding of the whole system of gas transference within the body. The gas depot method of investigation of gas tensions in the tissues is the only one which has yet been devised to give satisfactory direct results. A more critical discussion of the relation of the gas depot to tissue fluid and tissues is desirable so that a true interpretation of the results obtained by this method may be made. It is the object of the present article to attempt to clarify the various factors mentioned.

The Physiological Relations of the Tissues to the Respiratory Cycle.

The term "respiratory system" has been used commonly in physiological discussions to denote the whole system of gas transference of an organism, extracellular and intracellular; the same term has been associated also with pulmonary ventilation. To avoid confusion, the term "respiratory cycle" is here used to denote the system of gas transference between the atmosphere and the capillaries in the tissues. The respiratory cycle thus includes three main factors: pulmonary ventilation, gas carriage by the blood and the circulatory system both general and local. The "hæmo-respiratory system" refers to the part played by blood in the respiratory cycle. Other factors in the respiratory cycle are referred to specifically.

Tissue Requirements as the Raison d'Être of the Respiratory Cycle.

The fundamental importance of the requirements of the tissues in regard to the respiratory cycle is shown clearly in a reference by J. Barcroft in a discussion on "The Call for Oxygen by the Tissues",[1] from which an excerpt is given below. Although the reference is not solely to gas tensions, the same order of importance applies.

> The classical work of Pflüger[(a)] on the combustion of living material settled for all time, it seems to me, the logical order in which the constituent processes of respiration should be treated. The issue before Pflüger may be stated in a few words. Is the quantity of oxygen taken up by the cell conditioned primarily by the needs of the cell, or by the supply of oxygen? The answer was clear, the cell takes what it needs and leaves the rest. Respiration therefore should be considered in the following sequence. Firstly, the call for oxygen; secondly, the mechanism by which the cell elicits a response, the immediate response consisting in the carriage of oxygen to the tissues by the blood and its transference from the blood to the cell; thirdly, in the background you have the further mechanism by which the blood acquires its oxygen. It is not the habit of writers on respiration to adopt this order, quite the contrary, but their reason for placing pulmonary respiration in the foreground of the picture is a purely practical one—pulmonary respiration is more evident, both to the eye and the understanding. I imagine they will not quarrel with me if I make an attempt to treat the matter in what appears to be its logical sequence and make some estimate of the call which the blood has to meet before entering into a discussion of how the call is to be met. The present chapter will deal with the following theme: "There is no instance in which it can be proved that an organ increases its activity under physiological conditions without also increasing its demand for oxygen."

Correlation and Reciprocity of Tissue Requirements and the Respiratory Cycle.

The transference of oxygen and carbon dioxide to and from the cells depends essentially on the relation of the tensions of these gases in the cells to those in the respiratory cycle—that is, the transference is dependent on pressure differences. The study of gas tensions in either the respiratory cycle or the tissues is therefore incomplete without correlation of the two factors.

It will be seen later that the gas tensions in any tissue are not regulated wholly by the tissue itself; changes in gas tensions in the respiratory cycle and changes in circulation exert a modifying effect. That is, there is some reciprocity between the gas tensions of a tissue and those of the respiratory cycle. This is in accord with the general economy of the living organism: the functions of individual parts are subject to the maintenance of the whole organism. Thus, the changing requirements of any tissue are supplied by variations in gas tensions in the endeavour of the organism to maintain its normal state.

In the interpretation of changes in gas tensions in the tissues it is usually necessary to differentiate between the primary changes in the tissues and the secondary changes imposed by the compensatory action of the respiratory cycle. This again emphasizes the necessity for correlation of the gas tensions in the tissues with those of the respiratory cycle.

The Physical Relations of the Tissues to the Respiratory Cycle.

The Gas Tensions of a Tissue as a Mean.

The term "gas tensions of a tissue" must be regarded as a concept similar to that of gas tensions of alveolar air. Both tissue and alveolar air represent an aggregation of many separately functioning units, so that observed gas tensions are only a mean of perhaps widely different tensions. In any tissue, the gas tensions of the various component cells no doubt vary from cell to cell, particularly according to their relation to the functioning capillary network, as indicated by the work of Krogh.[7] This is of great interest in regard to the finding in gas depots in human subcutaneous tissues of oxygen tensions as low as four millimetres of mercury. As will be shown, this observed figure represents an extracellular tension, so that the mean intracellular oxygen tension is probably lower still. If, as seems probable, some cells of the subcutaneous tissue are closer to functioning capillaries than others, there must be in those cells furthest from capillaries an oxygen tension of nearly, if not quite, zero.

Tissue Fluid.

The cells of the tissues are not in direct contact with the capillary network, but are separated from it by tissue fluid through which there must be a gradient of gas tensions. As pointed out by Campbell, the gas tensions estimated by the gas depot method are extracellular; that is, the tensions are those of gases in the tissue fluid, not within the cells. Although the distances concerned in the

passage of gases through tissue fluid are small, the finding of very low oxygen tensions in some gas depots, sometimes twenty or thirty millimetres of mercury tension below the venous oxygen tension, indicates that from capillaries to tissue fluid alone there may be a considerable oxygen tension gradient. Hence, the oxygen tension within the cells may be even lower to an appreciable extent. The fact that the carbon dioxide tensions in the depots in human subjects are generally close to the venous carbon dioxide tension indicates that the tissue fluid forms usually only a very slight tension gradient for this gas.

Just as a tissue must be considered to have mean intracellular gas tensions, so the tissue fluid must be regarded as having mean gas tensions which are extracellular.

The Tissues and the Respiratory Cycle.

A useful, although imperfect, analogy may be drawn between tissue fluid and alveolar air in respect of the part they play in gas transference. Both these entities are media for exchange of gases between capillaries and adjacent sources of gases; both are gas reservoirs between an active and a passive factor. The passive factors are the two halves of the hæmo-respiratory cycle, the arterial blood and the venous blood. Arterial blood is the passive carrier of gases at tensions imposed by alveolar air; venous blood similarly is a passive carrier of gases at tensions imposed by the tissues. At either pole of this passive cycle the blood comes into contact, through the media of alveolar air and tissue fluid, with active factors—namely, the regulated inspiration of atmospheric air at one pole and the metabolic requirements of the tissues at the other. The organism as a whole is represented by a multitude of these cycles passing through the various tissues, every cycle having the alveolar air and arterial blood in common. Through this common link changes in the activity of any tissue, if sufficiently great to overcome the regulating factor of alveolar air, may impose effects on all other tissues.

It is the arterial blood which is regulated to supply gases to the tissues at tensions most suitable for their requirements. Only through changes in the gas tensions of arterial blood can changes in activity in any tissue directly impose changes in gas tensions on other tissues.

Changes in gas tensions in arterial blood also affect the activity of the respiratory centre. As is well known, changes in carbon dioxide tension in the arterial blood affect the respiratory centre far more than do changes in oxygen tension, thus tending to maintain the tension of carbon dioxide throughout the body at a more constant level than that of oxygen.

There are also indirect methods by which the activity of a given tissue may produce generalized gas tension changes throughout the body. The most important of these are changes in general body temperature and changes in the acid-base equilibrium of the blood and the mutual displacing effect of carbon dioxide and oxygen in the blood. At the same time there may be changes in circulation through non-active tissues. All these changes act synchronously in physiological conditions. For instance, in exercise the first local effect must be a tendency to a rise in carbon dioxide tension and a fall in oxygen tension in the muscles. Preceding and accompanying exercise there are general compensatory changes in lung ventilation and circulatory rate, produced first by nervous stimulation from the cerebral cortex and later by changes in composition of arterial blood. If the local changes in gas tensions in the muscles, which are imposed upon the venous blood, are sufficiently great to overcome the compensatory changes in lung ventilation and circulatory rate, these changes will be reflected through arterial blood directly to other tissues. At the same time there is a tendency to a rise in body temperature and in acidity in the blood and a tendency to a decreased power of oxygen carriage by the blood and perhaps changes in circulation throughout the tissues of the body.

The respective parts played by these several factors will be discussed later, particularly with regard to the effects of exercise. It may be noted here that all the changes associated with metabolic activity tend to cause inverse alteration between carbon dioxide and oxygen tensions in the tissues.

The manner in which the activity of one tissue may influence gas tensions in another tissue is of fundamental importance in the interpretation of results obtained by the gas depot method.

Transference of Gases by Diffusion.

The mechanism of exchange of gases between blood, tissues and alveolar air appears to be that of simple diffusion from a point of high gas tension to one of lower gas tension.[12][13][7][5] In regard to carbon dioxide, Krogh considered that not only did diffusion suffice for transference of gases from capillaries to tissues, but that "the CO_2 pressure differences between any point in the tissue and the blood must, moreover, in all circumstances, be an absolutely negligible quantity". Further reference to this statement will be made in discussion of normal gas tensions in the tissues which will be correlated with those of the respiratory cycle.

The Significance of Gas Tensions in the Transference of Gases.

The transference of gases necessitated by tissue metabolism is essentially quantitative; a certain degree of activity in a certain mass of tissue requires the transference of certain volumes of carbon dioxide and oxygen per unit of time. The significance of the corresponding gas tensions in the tissues and in the respiratory cycle lies in the fact that the pressure difference between these two factors in part determines the volumes of gas which may be transferred per unit of time. It may be noted that the finding of a low oxygen tension in a tissue signifies the possibility of a high pressure gradient between capillary blood and the tissues, facilitating supply of oxygen to the tissue.

The gas-tension gradient between capillary blood and tissue cells is only one of the factors concerned in the transference of gases. Other factors concerned are the resistance of tissue fluid to the passage of gases (probably a fairly constant factor), the area of the surfaces through which gases may diffuse and the distance to be traversed by gases from capillaries to cells. These latter factors are known to be variable, dependent on the state of the functioning capillary network. The state of the functioning capillary network in a tissue is in turn dependent on the degree of activity of the tissue and forms part of the compensatory mechanism associated with metabolism. The significance of gas tensions in the tissues at different degrees of activity is thus complicated by the concurrent changes in local capillary circulation. A tissue cell which at rest may have a low oxygen tension, being at some distance from a functioning capillary, during activity may have a higher oxygen tension due to the opening up of adjacent capillaries[7] in spite of the primary tendency to a fall in oxygen tension. A clear distinction must be made between the primary and secondary effects of tissue metabolism.

Tissue Metabolism as the Determining Factor of Gas Tensions in the Tissues.

To complete this elementary review of gas tensions in the tissues it is necessary to correlate these tensions with the essential metabolic processes of the tissues. The phase of metabolism with which these gas tensions are concerned has already been shown in the reference to Barcroft's statement that "there is no instance in which it can be proved that an organ increases its activity, under physiological conditions, without also increasing its demand for oxygen"; to this may be added: "and without also increasing its output of carbon dioxide". It follows that the primary effect of tissue activity must be to raise the tension of carbon dioxide and to lower the tension of oxygen in the tissue concerned. This demand for oxygen and liberation of carbon dioxide by tissue metabolism and the meeting of these requirements by the respiratory cycle regulate the tensions of these gases in the tissues. There can be no doubt that of these two factors, tissue metabolism is primary and the respiratory cycle is secondary or compensatory in the regulation of gas tensions in the tissues which must be interpreted accordingly.

Campbell[4][6] has shown clearly that gas tensions in the tissues show uniformity, within limits, under standard conditions. This limited uniformity exists among different species of mammals, amongst different individuals of a species and amongst different tissues of an animal at rest. Within this range of limited uniformity, minor but definite variations exist; these variations are themselves fairly constant for any species, individual or tissue. Further, it may be shown that in any individual or tissue, different degrees of activity produce definite alterations in gas tensions in the tissues.

Both the uniformity of gas tensions in the tissues and their limited variations require explanation. It follows, from the conception of tissue metabolism as the primary factor in regulating normal gas tensions in the tissues, that explanation of these gas tensions in terms of the state of the respiratory cycle is inadequate.

Campbell[6] refers to the observation by Pantin[10] on the critical oxygen tension below which movement in marine amœbæ ceases (30 to 40 millimetres of mercury). The inference that the somewhat similar oxygen tensions in mammals is a survival from the marine amœboid stage of evolution suggests an explanation of the uniformity of gas tensions in the tissues. It is probable that in the first instance unicellular organisms were adapted to function at the gas tensions of the original external environment. Since this first stage of adaptation, the immediate environment of the tissues probably has retained these gas tensions as their normal functioning levels. The development of a multicellular body, however, with specialization in tissue function must have necessitated concurrent modifications of cellular activity and of gas tensions in the tissues.

The growth of the multicellular organism also required the development of a distinct respiratory system for the transference of gases between the cells and the external environment. To this passive function of the respiratory system was added the power of active modification of changes in the external environment to meet the requirements of the tissues, thus enabling the maintenance of the original conditions of cell metabolism.

A similar explanation of the significance of the inorganic constituents of blood plasma has been put forward by Bunge[8] and Macallum[8][9] and commented on by Bayliss.[2] Some of these inorganic elements of blood plasma exist in proportions very close to those found in sea water. It has been suggested that this similarity is due to the perpetuation through heredity of the conditions of the original marine external environment of the first living cells. Macallum suggests further that the cells of the tissues may continue to reproduce these original conditions within themselves as well as in their immediate environment. It is not unreasonable to suppose that the cells of the tissues have perpetuated similarly the gas tensions of their original marine external environment.

The consideration of the probable evolutionary development of tissues in regard to cellular activity and gas tensions gives an explanation both of the uniformity of gas tensions in tissues and of their variations in different tissues under normal conditions. The uniformity appears to be a modified reflection of the original external environment of unicellular organisms, perpetuated by the respiratory system. The variations appear to be dependent on the development of specialized tissue functions with the corollary of different degrees of tissue metabolism. It may be emphasized that the cellular environment, which has become known as the "internal environment" of the organism, as part of the respiratory system, is regarded as subservient

to tissue requirements. The modifying effects which the respiratory cycle exerts on gas tensions of any tissue are directed to the maintenance of normal conditions both in that tissue and in all the other tissues which form the organism.

The explanation given of different levels of normal gas tensions in different tissues applies equally to the gas tensions in any particular tissue associated with different degrees of activity; that is to say, the levels of gas tensions are an indication of the degree of metabolism. However, the significance of differences in gas tensions between two tissues at rest varies from the significance of differences between activity and rest in any particular tissue. In the case of different tissues at rest the inherent differences of metabolism are permanent and as such must be fully compensated by the respiratory cycle. Thus, if of two tissues, such as the peritoneum and the subcutaneous tissue of a rabbit, the peritoneum has a higher normal oxygen tension than the skin, it may be regarded as having a higher metabolic activity; that is, the gas tensions of the peritoneum are adjusted permanently to compensate for a greater demand for oxygen. But in the case of varying degrees of activity in one tissue, the differences are temporary. A primary change of increased activity may be shown first by a rise in carbon dioxide tension and fall in oxygen tension. The compensatory changes of the respiratory cycle will tend secondarily to minimize or reverse these primary metabolic changes before normal resting levels of gas tensions are regained. It is possible that the changes in gas tensions noted at different levels of activity of a tissue may not be merely a temporary lack of compensation, but a temporary optimal condition. This would appear to apply particularly to the compensatory phase of the reaction to changes in metabolism.

The Gas Depot Method of Estimation of Gas Tensions in the Tissues.

The gas depot method of gas tension estimation has been discussed by Campbell,[6] and details of a technique in this method, suitable for both animal and human investigations, will be given in a later article.

The Relation of Gas Tensions in the Tissue Fluid to those within the Cells.

As pointed out by Campbell, the gas depot in the tissues has as its immediate environment a layer of tissue fluid, so that it is with gas tensions in this fluid that the gases in the depot are in equilibrium. In the discussion on the relations of tissue fluid to the tissues and the respiratory cycle it was seen that tissue fluid is a medium of gas exchange possessing gradients of tension for gases passing through it.

There are two groups of gas tensions to which the tissue fluid is exposed: those of the capillary blood and those of the tissues. Gas tensions of capillary blood are unknown and must represent a gradient from gas tensions of arterial blood to those of venous blood, so that tissue fluid may be regarded as exposed to tissues on the one side and to arterial blood and venous blood on the other. There is no indication from an anatomical viewpoint as to which of these groups will most influence the tissue fluid or as to whether gas tensions in the tissue fluid will represent a mean.

It is known that gases in alveolar air and arterial blood are in approximate equilibrium, so that it might be supposed by analogy that gas tensions in tissue fluid will be similar to those of the blood leaving the tissue fluid, that is to say, in this case, the venous blood. However, observations on normal human subcutaneous tissues occasionally give figures such as 55 millimetres of mercury for carbon dioxide tension and five millimetres of mercury for oxygen tension. The oxygen tension within the cells cannot be less than zero, so that the pressure difference between tissue fluid and the cells cannot be more than five millimetres of mercury in the instance given. Since the diffusibility of carbon dioxide through the tissues is about 25 times as great as that of oxygen, the intracellular tension of carbon dioxide must be almost identical with that of the tissue fluid as measured in the gas depot. It would appear that gas tensions in gas depots represent very closely the gas tensions within the cells.

Reactions of the Tissues to the Gas Depot.

Campbell has shown clearly that following an initial injection of gas into the tissues there is a sterile hyperæmic reaction lasting several days. This is similar to the well-known "foreign body reaction". It is difficult to determine the exact nature of this phenomenon; it has been found to be independent of the nature of the gas injected and its volume, temperature and water-vapour content. It will occur in the peritoneal cavity of the rabbit with volumes which could hardly cause appreciable tissue damage. I have noted this reaction after injection into a rabbit's peritoneal cavity of 20 cubic centimetres of gas which had just been withdrawn from the peritoneal cavity of another rabbit where gas had been present for weeks. The withdrawal and injection were made with a syringe warmed to body temperature and moistened with normal saline solution. It can only be supposed that the molecular nature of free gas as opposed to gas in solution exerts some stimulating effect, either directly on the tissues, or more probably, on the blood vessels.

After the acute hyperæmic stage of reaction there follows a quiescent stage in which the site of injection becomes thickened by a fibrous and fibrinous deposit. This is easily demonstrated in the rabbit in microscopic sections, as indicated by Campbell. In the human subject the thickening of the skin and subcutaneous tissues is more pronounced than in the rabbit. Thickening commences at once, but is usually only slight during the first seven to ten days. Thereafter the thickening increases rapidly until usually at the end of fourteen to twenty days the depot must be abandoned. This thickening, when present to a

great degree, is very similar to a non-suppurative cellulitis; but the temperature is not increased, nor is the part more than slightly tender. If a depot is allowed to come into this state and injections are then discontinued, the subcutaneous mass gradually disappears; but the skin remains slightly thickened and tough for many months, owing probably to the deposition of fibrous tissue.

It is obvious that the tissues are not in a perfectly normal state when gas is present. This abnormality is one of the shortcomings of the gas depot method. To what extent this abnormality affects the normal gas tensions in the tissues is unknown; but it is probable that the effect is only small. The most pronounced feature of the period of reaction in the tissues to an initial injection of gas is the rise in oxygen tension above normal. This gradually drops to a fairly constant level. In this period of constant gas tensions occasional observations in rabbits and many in human subjects give oxygen tension figures under 10 millimetres of mercury. It is obvious that the hyperæmia which is the essential feature of the initial reaction to gas injection must have subsided, or if present must be a negligible quantity. There is, however, a possibility that after the initial hyperæmic reaction there may be a reverse change of diminished circulation as evidenced by the deposition of fibrous tissue. This might tend to give low oxygen tensions. But if such a condition of diminished circulation interfering with gas tensions in the depot did exist, there would probably be a great delay in the response of the depot to changes in gas tensions in the respiratory cycle. Yet it is found that the gas depot is remarkably sensitive. Changes in respiration, for instance, sometimes cause appreciable changes in gas tensions in a subcutaneous depot at the end of a minute.

It appears fair to conclude that the gas tensions in the gas depots are a close reflection of gas tensions in normal tissues. It would be of great interest, however, to have comparative figures for gas tensions in the tissues by some other direct method.

Lag Effect in the Depot.

When gas tensions in the tissues alter rapidly there is some delay in the reflection of these changes in the gas depot owing to its volume, somewhat similar to the inertia of mechanical recording apparatus. This will be noticeable particularly in gas depots in rabbits, in which the volume is large compared to the surface of the depot, but will be of considerably less extent in depots in human tissues where the gas is a thin loculated layer. This lag will affect oxygen more than carbon dioxide because of the slower rate of diffusion. In terms of graphic representation, the crest of the curve denoting gas tension changes in a gas depot will tend to be lower and later, particularly for oxygen, than the actual changes within the cells.

Interpretation of Gas Tensions in any Tissue in Relation to the whole Organism.

In the discussion on the physiological relations of the tissues to the respiratory cycle it was shown that the activity of any tissue may cause changes in gas tensions of distant tissues. Tissues which may be used conveniently for the formation of gas depots are limited to the peritoneal cavity and subcutaneous tissues in rabbits and to subcutaneous tissues alone in man. The interpretation of observations in gas depots must differ according to the site of the experimental stimulus. This gives rise to the necessity for the consideration of different types of experiments which fall into three groups: first, experiments on subcutaneous tissues themselves, in which the tissues surrounding the gas depot are the origin of changes in gas tensions—for example, application of local heat; secondly, experiments primarily involving distant tissues—for example, exercise, in which cases the tissues surrounding the gas depot merely reflect changes in the respiratory cycle and indirect reactions of distant tissues; thirdly, experiments involving the body as a whole—for example, alterations in respiration, in which the tissues surrounding the gas depot may be taken as representative of the whole organism.

In the discussion of gas tensions in normal resting tissues consideration will be given to the relation of the tissues under observation to other tissues of the body.

Summary.

A discussion is given of the physiological and physical relations of the tissues and tissue fluid to the respiratory cycle and of the relation of gas tensions in the tissues to tissue metabolism. A survey is made of the gas depot method as an indication of gas tensions in the tissues.

Acknowledgements.

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THE TREATMENT OF OBESITY IN GENERAL PRACTICE.

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> Whatever may be the quantity that a man eats, it is plain that if he is too fat, he has eaten more than he should have done. One man may have a digestion that consumes food better than common, but it is certain that solidity is encreased by putting something to it.
>
> Samuel Johnson.

Obesity is not so much a disease as a constitutional habit or "diathesis". Its incidence varies to a certain extent with the fashions; by the slim-figured *demi-monde* of the present day the fashionable rotundity of "Boule de Suif" and other Maupassant characters would be classed as gross obesity. Throughout the ages fatness has always been popularly associated with good nature and mental sluggishness. Shakespeare's greatest figure of fun was a man of enormous girth, Falstaff. The poet put into the mouth of Julius Cæsar a wish to be surrounded by fat men, who do not think too much—"Sleek-headed men, and such as sleep o' nights". Shakespeare elsewhere made mention of what is now popularly known as the "middle-age spread", in his reference to the "fair round belly" of the later ages of man.

The popular mind also associates obesity with over-eating and physical sluggishness. That there is truth in this is proved by a glance at the celibate clergy, of whom an undue proportion are overweight; they tend to combine over-eating and physical ease with great mental activity. G. K. Chesterton, the most famous fat man of the present age, was a profound thinker and a man of quick wit; and St. Thomas Aquinas, the great mediæval philosopher, had such an immense abdominal protuberance that he could not sit at table unless its edge were carved into a concavity. Dr. George Cheyne, an eighteenth century English author, who weighed 448 pounds, was successful in reducing his bulk to normal dimensions by diet control.

The general notion that fat people do not live so long as thin ones is borne out by statistics, and all actuaries and assurance societies recognize that an obese proponent is not so good a risk as a thin one. This is supported by clinical experience, which readily recalls fat patients who put up only a poor showing against pneumonia or heart failure.

In country districts a great diversity of types is not met with, and it will be seen that nearly all of the cases reviewed were of a similar type. This, however, does not preclude valuable lessons being drawn from a study of a series of cases, even without the services of an expert pathologist or profound biochemical research.

Causation and Pathology.

As a guide to the treatment of obesity, it is advisable to consider in some detail its causation and pathology.

Obesity presents itself to the eye as the manifestation of an excessive deposit of body fat. The neutral fat which is stored in the subcutaneous tissues subserves several important functions. It is a readily stored, easily assimilable source of energy, of high caloric value; it is the chief energy reserve of the body. In addition, the layer of fat covering the body helps to preserve the body heat, and improves the æsthetic appearance by smoothing out irregularities. The sources of this depot fat, according to Samson Wright (1936), are: (i) the fat of the food, which is absorbed from the intestine and transferred directly to the depots; (ii) the carbohydrate of the food, transformed into fat by a process the details of which are unknown. This process is somehow linked up with body metabolism and the utilization of oxygen. A substance rich in oxygen is transformed into one poor in oxygen, and thus a good deal of oxygen is left over to aid metabolism. (In other words, the respiratory quotient is high.)

When the need arises, the depot fat is taken up again and utilized by the tissues. It is known that it is directly burned up; but here again the details are unknown. It is certain that the combustion can be carried out completely only in association with the combustion of sugar. If fat is incompletely burnt "acetone bodies" are produced, and this leads to ketosis.

The storage of fat can take place only if the caloric value of the food exceeds the energy requirements of the body. If this storage becomes excessive in amount, we say that the subject is obese. But obesity does not lend itself to accurate definition, for there are wide limits between which the patients (mostly female) complain of being too fat. As a rule the patient herself diagnoses her complaint and comes to the doctor to be made thin; only occasionally has the original complaint been of other symptoms, for which it was necessary first to treat the obesity.

Obesity in the past has been regarded as a symptom rather than as a clinical entity or syndrome; but advanced opinion is now commencing to swing round to the opposite view, that the underlying metabolic peculiarity is the same in all cases of obesity, though the immediate causes may vary (Gardiner Hill, 1935). Obesity is a disorder of metabolism, of the unknown mechanism which tends to keep the body weight stationary. The digestive and absorptive capacities are not excessive, for in the normal alimentary tract the assimilable portion of the diet is completely absorbed (Wright). Therefore there must be an abnormal capacity for storing food.

It has been customary to classify obesity under two main heads, exogenous obesity and endogenous obesity. (*a*) In exogenous obesity food is stored because of excessive supplies from without, or more simply, because of over-eating. (*b*) Endogenous obesity is caused by a lessened expenditure of energy due to a deficiency or disorder of the mechanism by which excess is eliminated. This mechanism is frequently said to be the endocrine

system; but there is no really conclusive evidence for this belief, except in so far as many metabolic activities are known to be regulated by endocrine glands, and in many instances obesity is accompanied by obvious endocrine disorders.

True exogenous obesity is not common, though it is easy to recall individual cases in which the increase in weight was correctly ascribed to a lately acquired bad habit of over-indulgence at table. It must be true that long-continued over-filling of the stomach will tend to cause the increased deposition of fat. But, no matter how much one over-eats, the body tries to get rid of the excess unless there is an underlying tendency to store food. In the normal subject there is a distinct inertia, or tendency for the body weight to remain stationary. If excess food is supplied, it is burnt off; if the body is starved, the metabolic rate falls in an attempt to keep the body weight normal (Wright). Thus in nearly all cases of obesity the endogenous factor is the chief one; though the exogenous factor must also play its part. For, no matter how disordered the mechanism may be, no person can become obese without taking food, and no fat patient can maintain his weight if he starves. But few obese patients grossly over-eat; most of them take a normal diet, little in excess of their caloric requirements. This slight excess, however, is not disposed of in the normal fashion. Where this abnormality rests is as yet unknown. It has been suggested by Bulger (1936) that the disorder resides in the adipose tissue itself. It is certain that there is no lowering of the basal metabolic rate. Sir Edmund Spriggs (1935), Keeton and Bone (1935) and Lyon (1938) recently reported large series of cases in which the basal metabolic rate was almost invariably within normal limits.

There is a good deal of circumstantial evidence to suggest that the "elimination of excess" mechanism is connected with the endocrine system.

1. Myxœdematous patients tend to gain in weight, and excess of thyreoid causes loss of weight. We know, however, that a lowered metabolic rate, pathognomonic of hypothyreoidism, rarely occurs in obesity. Moreover, myxœdematous patients are not invariably fat, nor are the deposits in myxœdema composed of normal adipose tissue (Wright).

2. The occurrence of obesity in connexion with lesions of the pituitary gland is far more suggestive: (*a*) In Cushing's adreno-genital syndrome, obesity is invariably seen with hypogonadism and hirsuties (Gardiner Hill). The lesion nearly always is a basophile adenoma of the pituitary; but occasionally a growth of the adrenal is found. (*b*) In other pituitary lesions adiposity is commonly present, though not invariably so (Wright). In gonadal deficiency due to hypopituitarism, the patient may be thin. In Fröhlich's adiposogenital syndrome and Dercum's disease varying lesions may be found—for example, a tumour or hæmorrhage of the posterior lobe of the pituitary, or similar affections of the hypothalamus (Hess and Kunstatder, 1938). Obesity sometimes follows encephalitis affecting the hypothalamus. Gardiner Hill states that great variations in fat deposition occur even with almost identical pathological lesions. Therefore, he states, the obesity-governing mechanism is independent of the endocrine disorders, except in Cushing's syndrome.

3. Obesity occurs frequently in children in connexion with genital hypoplasia. Hunt (1939) recently investigated a series of these cases, and his conclusions were that all these children grossly over-ate; that the obesity was purely exogenous, and that the genital hypoplasia was directly caused by it. He advanced convincing evidence of cures by diet of both obesity and genital hypoplasia. However, the weight of evidence in the majority of cases suggests that the hypogenitalism is not caused by the obesity, but that both are due to a common cause.

4. The frequent occurrence of obesity at the menopause, during pregnancy and after childbirth, when endocrine adjustments are taking place, suggests an intimate connexion with the glands of internal secretion. However, obesity occurs at other times also, and again the weight of evidence suggests that the obesity is a more or less casual by-product.

5. Diabetics can be fattened by large doses of insulin combined with a high carbohydrate diet (Bulger). (This, by the way, is a true exogenous obesity.) But the carbohydrate tolerance is normal in the average case of obesity. Spriggs reports a large series of cases to prove this. There was no case of glycosuria in my series.

Obesity is linked somehow with an abnormality of the water-salt storage, which is connected with the endocrine system. The water-balance is disturbed in Fröhlich's and Cushing's syndromes, and in myxœdema (Gardiner Hill). In the latter it returns to normal when thyreoid medication is introduced. The subcutaneous deposits of fat contain, of course, a large proportion of water. The adiposity of children tends to revert to normal at puberty or adolescence, while the obesity which follows an illness tends soon to a spontaneous cure. Sex has considerable influence on fat deposition, since it is very much more common in females. An additional factor here perhaps is the lessened physical activity in the female sex. Gravity has a slight influence on the distribution of the deposits, especially in old people, who are sometimes seen to be thick-set below the waist and slim above it (the "spreading" type). Race perhaps has some influence, a well-known example of racial adiposity being seen in the Levantines. (Perhaps, however, this is exogenous, due to the excessive indulgence in olive oil.)

Heredity is the biggest factor in determining the incidence of obesity. I have no statistics to prove this, such an everyday fact requiring nothing more than a casual glance at the families of one's acquaintances. Fat people tend to beget children who will become fat. Many of the more obese patients in my series had fat mothers, though this is by no means invariable.

Exercise has only a small influence on obesity. In theory, additional exercise should cause the burning-off of an appreciable amount of fat; but in practice its effect is almost negligible. Sluggishness in a patient does not necessarily lead to adiposity. Many fat people are quite active, while many thin ones are sluggish. But fat people frequently become sluggish.

Let me summarize the foregoing analysis of the causes of obesity. Probably the main factor is an underlying metabolic peculiarity, often hereditary, which is the same in all cases of obesity. In different cases, however, different immediate causes are found which bring this peculiarity into evidence.

The Types of Patients.

Obese patients come to the doctor for the relief of various symptoms, but the commonest reasons are æsthetic. The patient simply complains of being too fat, and wants something to cure it. (She usually expects a drug or a mixture that will make the fat vanish as if by magic.) She wishes to regain her girlish figure, and in most cases she would do anything (except forfeit the pleasures of the table) in order to achieve that end.

In all, I have dealt with 52 obese patients during the past three years. The large proportion complained of fatness pure and simple, while several of them came for treatment of different presenting symptoms, which may or may not have been connected with the obesity. Shortness of breath is a common symptom, and another surprisingly frequent concomitant is muscular pain. Shifting myositis occurred in several of the very fat ones, and its incidence appeared to decrease when the obesity came under control. Osteoarthritis of one knee was the outstanding trouble in two cases, while the symptoms of hyperpiesia (confirmed by examination) occurred in four. Strange to say, in no other case was the blood pressure raised above normal limits; and no case of heart failure was seen. One rotund girl, aged nineteen years, who had been frankly over-eating, complained chiefly of *acne vulgaris* (see Case V). Only four males were treated, including a feeble-minded boy, the subject of a congenital pituitary dystrophy, with whom I could do nothing. Of the others, one alone complained of obesity; one was suffering from gout, and the other from abdominal pain, which was found to be caused by diverticulitis. This man's symptoms were relieved within a week of his commencing to diet, and in that week his weight fell from fifteen stone to fourteen stone nine pounds. The paucity of males is explained largely by the fact that obesity is much more common in females; but it appears also that males are less likely to object to fatness and to seek its amelioration.

Two women suffered from gall-bladder disease, and both subsequently underwent operation. Two patients with myxœdema were seen, neither being grossly overweight. Both were treated with thyreoid only, with complete relief. One lost no weight whatever, and the second a few pounds only. Two patients with pituitary dystrophy were seen, both feeble-minded children whose mothers were non-cooperative; and nothing was achieved for either of them.

Forty-seven of the 52 patients were adult females, whose ages ranged from nineteen to fifty-six years. Six were unmarried young women aged under twenty-five years; one was a married woman who had not borne children, and the remainder had borne varying numbers of children, from one to eight. Several were seen at or shortly after the menopause, but the majority were in the decade from thirty to forty years. Nearly all of those who weighed more than fifteen stone belonged to this group. All the younger women who complained of obesity were worried about a commencing spread of figure, and the weight of few of them exceeded thirteen stone. The heaviest woman weighed sixteen and a half stone, and the lightest nine and a half stone. Four weighed over sixteen stone and six more over fifteen stone, while five weighed less than ten stone. Most of the younger women associated the rotundity with a pregnancy. All were active women doing housework, more than half of them being farmers' wives. All (except the girl of nineteen whom I have mentioned) stated that they were small eaters. Careful questioning confirmed this in most cases, or rather it established the fact that they were not gross over-eaters. Nearly all betrayed a disproportionate liking for bread, cakes and pastry, to the exclusion of meat, fruits and vegetables. This, however, is a fault common to most communities in Australia. Most of the very fat ones complained that they were becoming sluggish; obesity induced a lazy habit, rather than the reverse.

The patients were subjected to a routine physical examination, including the blood pressure and the urine. In no case was associated disease of any importance discovered except when its symptoms were the main cause of the complaint.

"Too much fat", says Sir Edmund Spriggs, "restricts movement, infiltrates important tissues, interferes with the action of essential glands, and makes the person less capable generally." But reasonable plumpness, he says, is good, and the fatness of convalescence, of pregnancy and of middle-age is good if not carried to extremes. In most of my cases the patient was the judge who decided when a cure had been effected; except in extreme cases the fight was waged on æsthetic grounds only.

The Treatment of Obesity.

The obvious rules to apply in the treatment of obesity are two: (i) reduction in the intake of food, (ii) increase in the output of energy. A rapid reduction could be achieved by complete fasting. In fasting, the tissues draw first upon stored carbohydrates, and later upon stored fats (Wright). But it is infinitely better to supply the basic needs of the body in proteins, vitamins and minerals. Out-and-out starvation is bad.

I limited my treatment in uncomplicated cases of obesity to the first rule. To increase the output

of energy of a normally active person is difficult, while for a sick patient it may be undesirable.

I have kept before me the truism that no person can gain in weight unless he is ingesting food in quantities more than enough to satisfy the needs of the body. If a quantity less than this is given, it requires only a simple mathematical calculation to show that the patient must lose weight. But the body practises economies when the food intake is lowered, and it is necessary to go far below the normal maintenance diet to effect a noticeable fall in weight. It can therefore be taken as a maxim that a sufficient reduction of food will cause the body weight to fall. In the opinion of Donald Hunter (1935), even the grossest cases of endocrine obesity can be treated by diet alone.

Bearing in mind these principles, I treated obesity in every case by diet alone. There is no other antidote. The direct antidote to the deposition of excess food is to restrict the supply. Thyreoid extract should never be used, as its use is not in conformity with good physiological principles. It has no direct effect on the fat deposits; it reduces obesity only by stimulating all the processes of metabolism, and inducing hyperthyreoidism, a pathological state. I have had no experience of its use for true obesity, and so I can give no personal accounts of its dangers. In moderate doses there would not appear to be any danger associated with the use of thyreoid extract; but it necessitates strict medical supervision, with a careful watch for symptoms of overdosage. Furthermore, some restriction of diet is necessary if the patient is not to be permanently dependent upon thyreoid extract; if the patient has to learn the principles of control by diet at last, she should instead be made to learn them at first. Thyreoid extract also is said to stimulate the appetite, and its effect in acting as a whip to drive many of the organs of the body is most undesirable for any patient, to say nothing of those past middle-age. In a word, thyreoid extract is unnecessary and its use is unscientific.

Dinitrophenol has been recommended by some writers. It is said to accelerate catabolism by its specific effect upon the tissues themselves (Dunlop, 1938). However, it is known to cause toxic manifestations, and cataracts have been said to follow its use (Dunlop; Gutzeit, 1937). It has not been used by the present writer.

The rate of loss of weight can be varied according to the degree of starvation induced. There can be no failure with adequate control of diet. The degree of success in treatment varies with the strength of the patient's resolve to forgo the pleasures of the table in order to become thin.

The basal needs of the body can be calculated according to the area of the body surface. The loss of heat varies as the surface of the body (Wright). A committee of the British Ministry of Health (quoted by Wright) lays down 2,700 Calories as the minimum need of a woman carrying out the duties of an average housewife. If the patient is given a diet of daily value of 1,000 Calories, the body weight must fall. A value of 1,500 Calories is uncertain in its effects (Lyon, 1938) on account of the economies practised by the body when it is in a state of privation. Little attempt was made to restrain rigidly the protein consumption, though the carbohydrate and fat contents of the diet were strictly controlled. Protein is regarded by Wright and other physiologists as having a specific dynamic effect on metabolism. Keeton and Bone (1935) do not confirm this, though they have been able to vary the protein intake between limits of 90 grammes and 13 grammes per day without any perceptible alteration in the rate of reduction in weight. Protein, therefore, seems to exert a certain "anti-fat" effect. If, as physiologists unanimously agree, most of the amino-acids are converted quantitatively into carbohydrate and urea, it seems certain that the carbohydrate produced from protein cannot be transformed into fat and stored away (Wright). The experience of the writer is that large amounts of lean meat can be taken without increase in the body weight; but the slightest relaxation with carbohydrate causes an immediate gain in weight, or at least a cessation of the downward trend.

This observed fact offers a clinical proof of the conversion of carbohydrates into fats in the body. The storage of carbohydrates as such has little to do with the control of obesity. Carbohydrate is stored as glycogen; normally 500 grammes are stored in the body, half in the liver and half in the muscles (Wright). In starvation the liver glycogen disappears; but the muscle glycogen is reduced only when the muscle is exercised. When a patient is receiving treatment for obesity, the glycogen in the liver is never completely exhausted, for fresh supplies are constantly being made from tissue protein, though this form of carbohydrate is not stored in large quantities (Wright).

All the patients in this series were weighed and given a diet containing ample fruit and vegetables, rich in protein, but low in carbohydrates and fats. They immediately began to lose weight, presumably by oxidation of the fat and the release of water in the subcutaneous tissues. No restriction of fluids was made; but in some cases it was necessary to prohibit the consumption of alcoholic liquor. Alcohol has an intrinsic food value of 5·6 Calories per cubic centimetre (Lawrence, 1933). Two ounces of pure alcohol, however, are the limit that can be oxidized by the body in twenty-four hours. This is equivalent to about three and a half ounces of whisky. Thus, if Lawrence's contention is correct, potations of even a moderate spirit drinker may represent a food value of 400 to 500 Calories per day. In addition to the alcohol, however, all the ales, sweet wines and liqueurs contain considerable quantities of sugar. It will be seen, therefore, that the latter must be rigidly prohibited. It is my custom to prohibit all alcoholic drinks at the start, and to allow limited amounts of spirits or dry wines after some progress has been made.

The diet was based on that of A. H. Douthwaite (1934); but certain modifications were made in the

course of time which did not appear to diminish its effectiveness. The caloric value of the diet is 1,000 to 1,100; but a certain amount of latitude has been allowed with proteins in the form of lean meat and egg-white. The intake of egg-yolk is limited to one per day. Some carbohydrate is allowed in the form of fruit, and an almost unlimited quantity of green vegetables and of certain fruits gives enough bulk to allay the pangs of hunger. A printed card is given to the patient which reads as follows:

Breakfast: One egg and white of one egg (or 2 ozs. of lean meat or fish). Fruit portion (one apple, one orange or its equivalent).

Lunch: 3 ozs. lean meat or fish. ½ pint vegetables. Fruit portion.

Dinner: Same as for lunch.

In addition, 1½ glasses skimmed milk daily. A cup of tea or coffee allowed at any time, with skimmed milk and no sugar. No fried foods. No sugar. No alcoholic liquor. No butter, cream or fat. No olive oil. No foods made from flour—as bread, toast, pastry, cakes, scones, puddings. No jam. No oatmeal. No sweets. No bananas, grapes, dried fruits, prunes. No potatoes, carrots, parsnips, onions, beetroot, peas, broadbeans.

A little variety may be used in preparing your meals, so long as the above amounts in 24 hours are strictly adhered to. Fruit may be raw or stewed without sugar; tinned fruits may be eaten if first rinsed in water. Meat may be roasted or grilled, but it must be lean. Egg custards with skimmed milk are allowed, also fruit salads without sugar. Lettuce salads allowed, with a chopped hard-boiled egg (yolkless), and dressing which does not contain cream.

No bread substitutes are given, and no patients had to use saccharin in order to make up for the deprivation of sugar. The fruits and vegetables disallowed are those of a higher carbohydrate value (Lawrence). The caloric value of the diet is not especially calculated in every case. It will be seen by reference to the table (Wright) that its value is below 1,200 Calories. The therapeutic test proves it to be adequate in every case.

This diet will be seen to contain all the ingredients necessary for the bodily economy—plenty of protein, some carbohydrates (to help burn off the stored fat), a little fat, and plenty of minerals. The vitamins would be supplied by the raw fruits and vegetables, and the milk and the eggs. It is regarded as particularly important to impress upon the patient the necessity of avoiding certain foods like poison; but no great restrictions were put on the eating of fruit, except in the rare cases in which the patient failed to reduce her weight with this diet (all of these patients, however, were suspected of disobedience). Usually the patient is appalled by the prospect of having to abandon all her favourite foods; but great efforts are made to persuade her to try it for a fortnight. A substantial reduction in weight is faithfully promised, conditional, of course, upon her adhering rigidly to the diet. The rationale of the proceeding is explained, and "drugs" are definitely ruled out.

In many cases no insistence was laid upon the weighing of the ingredients, and the patient was in fact allowed to eat as much meat, fruit and vegetables as she desired. She was merely given a list of prohibited foods. In all cases reduction of weight resulted, though this as a rule was slower than when the amounts were restricted. This method was adopted with most of the patients who weighed less than ten stone; such a one is the patient in Case X.

With increasing experience, I am tending to put no limit upon the consumption of proteins, and lately I have almost convinced myself that fats need not be severely restricted. I feel that in the future it will be my rule to allow more latitude in both these directions.

The patient usually suffers keenly from pangs of hunger and weakness for a few days, but these pass off within two weeks. An immediate subjective improvement is noticed when these symptoms have passed off. She soon loses her craving for bread and sugar, and an increased activity and improved general health are noticed. These benefits frequently appear within the first fortnight, before the weight has been reduced to anything remotely approaching normal proportions. If the patient returns in a few days to complain of hunger and weakness, their early disappearance can be promised with mathematical certainty. The appetite for consuming large quantities of carbohydrate (which is a national vice) soon passes off if the patient makes up her mind about it. The patient returns after fourteen days, rather disconsolate, and says: "I'm still as fat as ever." When she stands on the scales, she sees that five to ten pounds (fourteen pounds in one case) have been lost, and immediately takes fresh heart. The problems of the successful treatment of obesity are almost solely psychological.

No recommendations were made regarding exercise, as all patients were normally active, and after losing a stone in weight they felt more energetic and took more exercise. Exhausting long walks have the undesirable effect of stimulating the appetite.

Historical.

The principles enumerated above in the treatment of corpulence have, of course, been well known for many years. Hutchison (1901) relates the story of William Banting, who was so fat that he could not tie his shoes, and he had to descend the stairs backwards. After many unsuccessful treatments, he was advised by an ear specialist named Harvey to abstain from bread, milk, butter, sugar and potatoes. So successful was this régime that Banting in his enthusiasm edited in 1863 "A Letter on Corpulence Addressed to the Public", in which he laid down the principles of weight reducing on perfectly correct lines.

Hutchison also quotes Dancel, a French military surgeon, who wrote on obesity in 1864. His dietetics were securely founded, and he mentioned amongst other things that alcohol should be avoided. Dancel and others were in error in restricting all fluids. The erroneous belief that restriction of drinking was essential in the treat-

ment of obesity passed out of court many years ago, though it still has wide acceptance amongst the public and even with some practitioners. Hutchison mentions many others who wrote on obesity before 1900, notably Ebstein, Herschfeld and von Noorden. He believed that carbohydrates were more likely than fats to lead to the deposition of adipose tissue.

Obesity, however, has for many years been the special domain of the quack and the unscientific practitioner. The public is avid for nostrums which will give short cuts to health, and avoids if possible the difficult path which enjoins the control of appetite. The discovery of the active principle of the thyreoid gland was thought by the profession at first to have provided such a labour-saving cure; but experience soon proved the falsehood of this assumption. Nevertheless, thyreoid extract is still widely prescribed by medical men as an antidote for pure obesity; and such standard text-books as that of Price (1929) have recently recommended its use.

Progress.

If the patient obeys instructions, she invariably loses five to nine pounds in the first fortnight, and a similar quantity in the second fortnight. If she loses only three pounds or less, it is regarded as almost a certainty that she has not adhered rigidly to the diet. It is found that the heavier patients almost invariably lose greater amounts than the less bulky ones, and that after six weeks or so most patients settle down to a steady loss of one and a half to two pounds per week. This is explained on the theory that, as the patient becomes thinner and partial starvation sets in, an automatic lowering of the basal metabolic rate takes place. The body begins to practise economies after a few weeks of lavish expenditure. It was my experience that the majority of the patients did not persevere. After a few attendances, having lost one or two stone, they were seen no more and returned to the fleshpots. My most determined patient was one who almost exactly halved her weight; reduction became an obsession with her and she kept setting herself fresh limits of slimness. Her record is as follows:

CASE I.—I.B., aged thirty-five years, was a nulliparous married woman. On October 15, 1937, she weighed 15 stone 10 pounds; on October 29 she weighed 15 stone; on December 23 she weighed 13 stone 8 pounds and on December 30 she weighed 13 stone 11 pounds. She then confessed to having eaten bread more than once, and rigid instructions were issued. On January 14, 1938, her weight was 13 stone 1 pound; on January 28 it was 12 stone 4 pounds; on April 1 it was 11 stone; on May 26 it was 10 stone; on November 24 it was 8 stone 10 pounds and on June 2, 1939, it was 7 stone 13 pounds.

This patient's enthusiasm knew no bounds. She made a keen study of her case, and is now able to regulate her weight almost at will. So sensitive was her metabolism to carbohydrates that the addition of only one slice of toast per day to the standard diet caused her weight to rise from 7 stone 12 pounds on July 21, 1939, to 8 stone 1 pound on July 28, 1939. This appears to support Banting, who regarded carbohydrates as "human beans"; that is, they cause deposition of fat out of all proportion to the amount taken. The patient was then persuaded to eat some pure fats over and above the standard diet. For several weeks she took fat meats, fried her food many times, and had a daily ration of a quarter of a pound of butter and three ounces of cream. Carbohydrates were again strictly interdicted. This regime was commenced on July 28, 1939, when her weight was 8 stone 1 pound. On September 4, 1939, she weighed 8 stone 3 pounds only.

It frequently happens that, after a steady loss in weight for several weeks, the rate of loss slows down to zero, and the weight becomes stationary before the full object has been attained. The patient's explanation is that she has "got used to the diet". Instead of ordering fresh privations, however, it is wise to question the patient in a detailed manner, when it will be revealed that she has begun to relax the régime. In some cases the patient will not admit it even under questioning, but I generally regard it as the more likely explanation. Some people find it very hard to confess to weaknesses of the flesh. It is perhaps wise in these cases to alow a little relaxation, if good progress has been made in the first few weeks. The progress then will be slower, but the patient will be happier. Here are some typical cases of failure to persevere:

CASE II.—F.B. was aged thirty years. On May 22, 1939, her weight was 16 stone 2 pounds; on June 2 it was 15 stone 10 pounds; on June 16 it was 15 stone 10 pounds. She admitted her failure to persevere after two weeks.

CASE III.—A.P. was aged fifty-six years. She complained of hot flushes and of shortness of breath. The record of her case is given in Table I.

TABLE I.

Date.	Weight.	Blood Pressure. (Millimetres of Mercury.)	
		Systolic.	Diastolic.
	st. lb.		
April 23, 1938	12 4	240	140
May 20, 1938	11 10	210	140
June 10, 1938[1]	11 10	190	120
July 1, 1938	11 5	200	130
August 29, 1938	11 0	230	120
October 3, 1938[2]	10 7	230	130
October 28, 1938	10 11	230	125
January 5, 1939	11 1	210	125
March 29, 1939	11 11	250	140

[1] Encouragement was applied at this stage.
[2] She refused to diet any more.

In this case the blood pressure decreased considerably with the first fall in weight, but did not continue to do so. Later this process was reversed. I do not think there is any real association between the blood pressure and the body weight, in spite of the cases quoted by Murray Scott (1939). Sir Edmund Spriggs, however, says that circulatory disturbances may be relieved by the decline in body weight. This patient, by the way, eventually obtained a good deal of subjective relief with œstrin. But this was parallel with an increase in weight as she returned to the fleshpots. (Therefore

the endocrine disorder must have been independent of the obesity.)

CASE IV.—J.S., a young unmarried woman, aged twenty-three years, suffered from chronic cholecystitis. On August 13, 1934, her weight was 12 stone 8 pounds; on August 30 it was 11 stone 13 pounds and on March 9, 1935, it was 11 stone 5 pounds. The patient was only spasmodically on diet. On April 3, 1935, cholecystectomy was performed, on May 25 her weight was 10 stone 12 pounds and on July 4, 1939, it was 13 stone 5 pounds.

Her appearance now is very gross; she has a coarse complexion and hirsuties She promised to try to restrain her appetite, but was unequal to the trial. Her efforts were successful in reducing her weight to 13 stone 1 pound on August 9. But out of all proportion to this loss of weight was the improvement in her sense of well-being and in the apearance of her complexion.

This is no isolated instance; it appears that the curtailing of the undesirable elements in the diet has upon the body a beneficial effect not entirely due to the slight loss of weight.

CASE V.—M.T. was a girl, aged nineteen years. She complained of *acne vulgaris* and had gained two stone in weight in four months. Hers was a case of frank over-eating. On June 13, 1938, her weight was 12 stone; on June 22 it was 11 stone 10 pounds; on June 30 it was 11 stone 9 pounds. At this stage she admitted her inability to control her appetite. On July 8 she weighed 11 stone 6 pounds and on August 8 she weighed 11 stone 11 pounds. She declined to diet any more.

CASE VI.—M.W. was a married woman, aged thirty years. She had grown fat after the birth of her second child. On December 12, 1938, her weight was 12 stone 13 pounds; on January 9, 1939, it was 12 stone 6 pounds; on February 7 it was 11 stone 13 pounds; on March 7 it was 11 stone 10 pounds; on April 29 it was 11 stone 11 pounds and on July 7 it was 11 stone 11 pounds. This patient considered that she had become used to the diet; but a more likely explanation is that she had never tried it fully.

CASE VII.—E.Y. was a woman, aged forty-four years. On June 8, 1934, her weight was 14 stone 7 pounds; on June 21 it was 14 stone 2 pounds and on July 5 it was 14 stone. At that stage, when questioned, she admitted that she had been to supper three times, listening to a broadcast description of cricket test match. On July 20, 1934, her weight was 13 stone 9 pounds; on August 16 it was 13 stone 6 pounds; on September 14 it was 13 stone 6 pounds and on November 8 it was 13 stone 6 pounds. When seen in the street lately she was very fat indeed, being probably in the fifteen-stone class.

An added difficulty in private practice is the unwillingness of patients, for reasons of finance, to pay frequent visits to the surgery. Here is a case in point:

CASE VIII.—P.D., aged twenty-eight years, had gained in weight after the birth of her first child. On October 29, 1936, her weight was 12 stone; on November 13 it was 11 stone 5 pounds. She did not return to the surgery, but continued to diet. When seen lately she was a slim young woman, in spite of the arrival of another baby.

Here is one of the more recent cases:

CASE IX.—Q.E. was aged twenty-five years. Her second baby had been born on March 16, 1939. On May 4, 1939, her weight was 11 stone 7 pounds; on May 15 it was 10 stone 12 pounds; on June 6 it was 10 stone 6 pounds and on July 5 it was 9 stone 13 pounds. She now considers that she is slim enough, and is allowed to have one slice of bread and butter with each meal. An interesting point concerning this case was the great improvement in lactation which occurred immediately when dieting was commenced. The baby, which had been making very slow progress, straight away began to gain weight at the rate of seven ounces per week, and has continued to do so.

This confirms the views of Samson Wright on lactation: (i) An increase of fat in the diet does not increase the yield of fat. (ii) An increase of protein in the diet causes an increased quantity of milk, and increases the total fat yield. (iii) A superabundant diet does not increase the yield unless the protein is increased.

Here is a similar case:

CASE X.—M.K. was aged twenty-nine years and her normal weight was eight stone. She complained that her figure had deteriorated after the birth of her fourth baby. When the baby was fourteen weeks old she weaned it, as her supply of breast milk had given out. Her diet was then restricted. On December 1, 1936, her weight was 9 stone 7 pounds; on December 12 it was 9 stone 4 pounds. Her milk supply returned; both breasts were full. On February 10, 1937, her weight was 8 stone 2 pounds.

This patient is keeping her weight stationary by a rigorous watch on the carbohydrate content of her diet. She does not restrict her intake of fats, but limits carbohydrate to one slice of bread at each meal, and eats no cakes at afternoon tea.

CASE XI.—I.F., aged twenty-eight years, who had four children, complained of shifting muscular pains, in addition to an increase in weight. On December 16, 1938, her weight was 13 stone 9 pounds; on December 30 it was 12 stone 12 pounds; on January 13, 1939, it was 12 stone 3 pounds; on January 27 it was 12 stone 1 pound and on March 13 it was 11 stone 5 pounds.

This patient still complains of myalgia, but feels greatly benefited.

Great subjective benefit was noted in nearly every case. Many of those patients who tackled the subject in only a half-hearted manner and lost only a few pounds in weight admitted that they felt much healthier and more active. In some cases, of which Case IV is an example, improvement in general health was obvious. Acording to Anderson (1935), the results of dieting in obesity are as specific as those of thyreoid medication in myxœdema. There seems to be no doubt that benefit results from the elimination of milled flour from the diet, and its replacement by animal proteins and fats, fresh fruit and vegetables. Experiments conducted by May Mellanby (Wright) and others prove that an excess of refined carbohydrates has a positively deleterious effect upon the tissues.

The patient in Case XII lost fourteen pounds in her first two weeks of dieting:

CASE XII.—E.H. was a very large woman, aged thirty-four years. On January 9, 1939, her weight was 16 stone 7 pounds; on January 23 it was 15 stone 7 pounds; on February 20 it was 14 stone 10 pounds; on March 8 it was 14 stone 5 pounds and on March 31 it was 14 stone 8 pounds. At this stage she admitted indulgence in luxuries. On April 24 it was 14 stone 3 pounds.

CASE XIII.—C.T., aged twenty-five years, was a young woman who was not really fat, merely plump. On March 8, 1938, her weight was 9 stone 11 pounds; on March 22 it was 9 stone 2 pounds; on April 5 it was 9 stone 1 pound; on May 10 it was 8 stone 11 pounds and on June 3 it was 8 stone 9 pounds.

Once again, the original good resolutions were not kept in their entirety.

As progress occurs, a certain degree of relaxation of the routine may be allowed. This usually takes

the form of latitude with quantity only, and the prohibited foods are still prohibited. In some cases, however, qualitative relaxations are allowed and the patient is permitted to eat restricted amounts of carbohydrates, such as a slice of bread with each meal. But care must be taken, for even small amounts of carbohydrate sometimes result in the renewed deposition of fat. In practice the restriction of carbohydrate generally causes the automatic restriction of fat, for a diet containing excess of unbalanced fat is nauseating.

Another factor which modified the routine was my estimate of the patient's intelligence and grasp of the essentials of the case. With the less intelligent, principles must be rigidly enforced. But, with one who understands her case better, such modifications as those already mentioned are readily allowed. This naturally causes the decline in weight to be much slower. It is frequently found, also, that the patient voluntarily adopts part only of the restrictions. Being intelligent, she can vary her weight at will by modifying the routine. Such a case is the following:

CASE XIV.—M.L. was aged twenty-five years and had one child. On May 4, 1939, her weight was 11 stone 13 pounds. She promised to carry out all instructions, but failed to reappear. When seen on August 9 she weighed 10 stone 6 pounds. She stated that from the start she allowed herself limited amounts of carbohydrate, which she varied according to progress.

Maintenance.

When the weight has been reduced to the desired level, how is it to be kept stationary? The self same principles are involved. The cooperation of the patient is essential; more so (if possible) than previously. Usually quantitative restrictions have been relaxed by this time, and the patient is not now required to weigh her food. If she continues to lose weight, more latitude is permitted by the judicious addition of carbohydrates. A slice of bread may be allowed at each meal, and the result noted after a week. But it is extremely difficult to persuade the patient to keep up her attendances at the surgery, and it is not unusual for her to lose ground at this stage.

My impression corresponds with the experience of Banting years ago, in that the ingestion of excess carbohydrates, even to the extent of only one or two indiscretions, is immediately punished by the renewed deposition of fat. It appears that the effect of indulgence in fat is not nearly so pronounced. (See Case I.) Ebstein, about 1880, advised his patients to restrict carbohydrates only. There is a good deal of evidence in favour of the tempting theory that the metabolic error in obesity consists of an excessive sensitiveness to the absorption of carbohydrate. It may be an abnormality of the mechanism which converts carbohydrate into fat.

To keep the body weight stationary at a low level, the exercise of a fair amount of intelligence on the part of the patient is required. Most patients by this time have a good understanding of their condition, and have seen how promptly all dietary indiscretions bring punishment. Those who have been successful in remaining slim have been largely left to their own devices, with indications as to how much of the various ingredients are allowed. No success is likely to be attained with stupid patients, either in reducing weight or in maintaining a reduction if by chance obtained.

Obesity in Children.

I have treated no child for obesity. I do not think treatment of children for obesity is at all necessary, except in the rare cases of dyspituitarism with distressing fatness. Most rotund children do not need attention. If gross over-eating is indulged in, the mother should be admonished to control it; but as most children over-eat if judged by adult standards, it is difficult to know where to draw the line.

Obesity in children usually becomes much less pronounced at puberty or adolescence. Fatness in children is often associated with sexual hypoplasia. This usually foreshadows a late puberty, which eventually arrives in all but definitely abnormal cases. Obesity in adolescent girls is frequently associated ith mild hirsuties.

In the grossest cases, such as those of Fröhlich's syndrome, treatment can be carried out only with the cooperation of the parents and relatives. A child cannot be expected to abstain, and it is very difficult for all concerned to attempt to control a child's diet. Given success in dieting, one could expect a cure in even the most advanced cases. Hess and Kunstadter recommend thyreoid for certain children, not definitely cretinous, whom they regard as suffering mildly from hypothyreoidism. I do not think that this procedure is at all advisable. As cretins are almost invariably underweight, obesity in a child should not suggest thyreoid hypoplasia unless it is accompanied by other evidence of a much more convincing nature. A more likely theory is that the pituitary hypoplasia, which is known to exist in many obese children, may lead to a deficiency of the thyreotrophic hormone. But the day should have passed when mere fatness led the clinician to think first of hypothyreoidism. Wharton (1939) says that underweight is as common as obesity in mild hypothyreoidism, no matter at what age.

Obesity and Associated Chronic Disease.

Coombs, Reader and Catlin (1937) give certain contraindications to dieting. These are as follows: (i) extreme old age, (ii) acute disease, (iii) any severe disease except heart failure, chronic nephritis and gout, and (iv) symptoms of circulatory or nervous instability.

No invariable rule can be laid down. Each case should be considered on its merits, and the obesity should be treated if treatment is not considered undesirable. According to Spriggs, the course of some chronic diseases is favourably influenced by the relief of the obesity, the presence of which in association with other diseases is usually quite accidental. Symptoms of circulatory instability may occur with the acute hunger which sometimes ensues in the first two weeks. I do not regard such

symptoms as a contraindication to diet control unless they are very severe. They cannot be due to starvation, as there is a good store of subcutaneous fat on hand.

As a rule, one should be chary of ordering exercise for patients suffering from chronic heart or kidney disease. A diet high in protein should not be prescribed in the presence of certain diseases, such as gout, in which it is contraindicated. The bulk of medical opinion would not favour such a diet in the presence of associated chronic kidney disease. Langdon Brown and Hilton (1936), however, quote many authorities to show that advanced opinion is moving in the opposite direction.

Many symptoms suggestive of heart disease, such as exhaustion and shortness of breath, disappear after the loss of a few stone in weight. Fat people should be dieted before being subjected to abdominal operation (that is, if the operation can be postponed with safety to the patient). As a general rule, however, associated diseases can be disregarded, if it is thought expedient to deal with the obesity.

Summary.

1. Obesity is due to an inborn metabolic abnormality, not identical with any known endocrine derangement. Perhaps this abnormality resides in the mechanism which converts carbohydrate into fat.

2. It is commonly hereditary, and is seen most frequently in females aged over thirty years.

3. Obesity can be treated in all cases by diet alone. The restriction of carbohydrate is more important than that of fat, while restriction of protein is hardly necessary at all. The use of thyreoid is not only unnecessary, but unscientific.

4. The chief difficulty in the treatment of obesity rests in securing the cooperation of the patient.

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WAR WOUNDS OF THE HEAD.[1]

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WAR-TIME surgery is largely concerned with the care of infected wounds and compound fractures, so that the general principles applicable to these classes of surgery always hold good. But wounds of special regions and special organs present their own particular problems as regards treatment and prognosis, and none more so than head wounds. Careful pre-operative study is necessary to determine the nature of the damage caused by the missile, and the extent to which treatment must be carried out, lest unnecessary harm be done at operation. This is accomplished by accurate neurological and radiological examination, and then by a rather more fastidious and time-consuming technique than is adopted with injuries in other less delicate regions.

Segregation of Casualties and Team Work.

The ideal method of dealing with these casualties is to segregate them in special hospitals, or at any rate to allot them to special teams with neurosurgical experience, as early as possible. Such a team should consist of a surgeon, a first assistant and an anæsthetist (skilled in the administration of "Avertin", and in the giving of blood transfusions and fluids by the intravenous route), a radiographer or X ray technician, an instrument attendant, a theatre attendant, and an orderly or male nurse (trained in the shaving of scalps, so that no fresh abrasions or lacerations are caused!). Adequate equipment and a supply of the correct instruments, dressings *et cetera* are essential too. Continuity of treatment is also advantageous, and arrangements should be made to hold the patient until he is out of danger and thus obviate all unnecessary movement. Such ideals may, however, not be attainable; and with the possibility of air-raid casualties occurring among the civil population or in isolated campaigns, as would probably happen in this country, it behoves every medical officer or practitioner to fit himself as far as possible to handle these cases; and this paper is presented with this object in view.

Classification of Head Wounds.

The wounds can be graded according to their severity (after Cushing) as follows:

(i) Scalp wounds.

(ii) Wounds with superficial damage to the skull.

[1] Read at a meeting of the Illawarra Suburbs Medical Association at the St. George District Hospital, Kogarah, on October 26, 1939.

(iii) Wounds with more severe damage to the skull, but without dural penetration.

(iv) Wounds with injury to scalp and skull, in which the dura has been torn.

(v) Wounds with indriven bone fragments, or retained foreign bodies, or both.

(vi) Wounds in which bone fragments or foreign bodies have reached the lateral ventricles.

(vii) Penetrating or through-and-through wounds of the head.

(viii) Penetrating or perforating wounds opening the mastoid air cells or nasal accessory sinuses.

Clinical Examination and Indications for Treatment.

The management of wounds of the head is not a mere exercise in operative technique. The earliest neurological signs must be carefully noted and checked, especially with regard to physiological defects proper to the injured area. Only in this way can true significance be assigned to an increase or change in signs later on, such as may indicate abscess formation. Positive signs may be present with the simplest looking injuries; they place the onus on the surgeon to satisfy himself thoroughly as to their cause, and not to aggravate them by his treatment. By these means can he decide whether to explore under a groove on the skull surface or a fissured fracture; and, more important still, whether to open the dura or not. This decision is very important, as the dura is a most valuable barrier against infection. Many wounded with head injuries who survive to reach the surgeon, have escaped death from the initial injury, have already regained consciousness (if indeed it was ever lost), but have still to run the risk of septic encephalitis, meningitis, osteomyelitis or abscess formation. If they are still unconscious, the outlook is much worse, and means that generalized as well as severe localized damage has been inflicted on the brain, or that some extensive intracranial blood clot has formed and needs evacuation. Thus, early operation is indicated (i) to eliminate or prevent sepsis by as complete excision of damaged tissues and *débridement* as is possible, with primary closure, and (ii) to relieve neurological symptoms and signs.

The power of the scalp, skull and brain of overcoming infection, once damaged tissues, foreign bodies and indriven bone fragments have been removed, is considerable. The worst cases are those in which there is a deep wound with a foreign body lodged in or near a ventricle, where sepsis can readily spread and cause an ependymitis. If operation in these cases is delayed more than twenty-four hours the death rate is much higher; and to avoid this delay, careful arrangement of transport and operating facilities is required.

First Aid and Pre-Operative Considerations.

Although rarely a serious factor in local wounds of the head, unless severe scalp bleeding has occurred, primary shock must be dealt with before any operative procedures are undertaken. This is accomplished by relief of pain and by warmth and the administration of fluids. Blood transfusion may be needed in severe cases, but usually only if visceral or limb injuries coexist. During this time hæmorrhage from the scalp should be controlled, and tetanic and gas-gangrene antiserum should be given in prophylactic doses; the patient should also receive two grammes of sulphanilamide or two grammes of sulphapyridine, or both, according to the infecting organisms under suspicion. Avoid morphine if possible; if the patient is restless or fully conscious, give half an ampoule of "Dial" or one-third of a grain of "Omnopon" before operation, but only after the clinical examination has been made. Then have an X ray examination made of the skull in all cases except those of scalp wounds only, to determine the presence, situation and number of fractures, indriven bone fragments and foreign bodies. Two pairs of stereoscopic, one pair of postero-anterior and one pair of lateral exposures are best; and the use of a Potter-Bucky diaphragm or a Lysholm grid is essential. Finally, the type of the patient's blood should be determined, in case transfusion is needed later, and then the whole head should be carefully shaved, in order to avoid inadequate exposure and to eliminate contamination and difficulties with subsequent dressings.

Operation.

Anæsthesia.

Local infiltration anæsthesia with 1% "Novocain" in normal saline solution, to each ounce of which are added three drops of adrenaline, is usually sufficient, if placed liberally under the skin and under the galea, to surround the affected area completely. This will render the bone and all other regions insensitive, except the larger meningeal vassels and the basal *dura mater*. For non-cooperative patients, "Avertin" in basal doses may be needed, and even ether in small amounts may be given by the intratracheal route in extensive procedures. A reliable syringe with well-fitting piston and attachable needles is a *sine qua non*.

Posturing and Draping of the Patient.

A thin sponge rubber mattress should be procured for the operating table, and the patient should be placed in the supine, lateral or prone position, according to the situation of the wound. In the prone position, some form of forehead and shoulder rests will be needed. In others, the head should be placed on a shaped sandbag, padded with rubber, and raised somewhat above the level of the body, to prevent venous congestion. The wrists should be tethered by manacles to the table, and a padded strap should be placed across the thighs, with a pillow behind the knees if the patient is supine, or under the ankles if he is prone. A support for the feet is also helpful. At this stage, needles should be inserted deeply into the muscles of each thigh, or else into a small vein of the foot or leg, through which saline solution with 5% glucose may be slowly given by continuous intravenous drip during the operation from a "Soluvac" container suspended two feet above the patient. A needle thus arranged also serves for a blood transfusion, if it is needed in a hurry.

An instrument tray should be fixed across the patient, to clear his chest by three or four inches, either as an attachment to the table or on a separate stand. The sterile towels, wrung out in a 1 in 500 solution of perchloride of mercury, can then be draped from the skull on to it; free access to the patient's mouth and free ventilation for a good airway are thus allowed. Air or oxygen may be blown under this tent-like arrangement. The drapings must be kept clear of the patient's face, otherwise he will feel as if he is being suffocated.

After the scalp has been "prepared" a final coating of Bonney's blue should be given, and the actual site of the

operation should be covered with a single piece of gauze, wet with a 1 in 500 solution of perchloride of mercury. The wet towels are more readily draped around this, and fixed in position to the scalp with sterile safety pins through wheals of locally anæsthetized tissue. The whole body is covered with a dry sheet, with an adjustable opening cut out at one end to give access to the wound. The necessary area is then infiltrated with local anæsthetic, and everything is ready for the operation to commence.

Wounds of the Scalp, with or without Superficial Damage to the Skull.

The edges of the scalp wound must be excised as cleanly, yet conservatively, as possible, care being taken to control fresh bleeding with digital pressure until artery forceps have been applied, at regular intervals of one centimetre, to the galea, which is thus pulled back over the bleeding edge. These forceps are gathered into bundles by rubber bands and held thus whilst a careful inspection of the bone is made; if necessary the incisions must be prolonged to permit opening up of undermined regions of the scalp. A painstaking *débridement* is then performed by "swishing" with warm saline solution from rubber ear-syringes, and sucking with a glass "sucker". Gauze "mastoid" strips and sponges are used for swabbing. The wound should then be closed with fine black silk sutures in two layers, the first picking up the galea, to control arterial bleeding and prevent blood getting under it. Tension along the skin edges must be avoided at all costs. One or more soft rubber drains should be left in position at the ends for twenty-four to thirty-six hours.

Sometimes a certain amount of plastic work is necessary to enable the edges to be drawn together; and the triradiate method of excision and incision is the best. Long tension incisions through the whole thickness of the scalp to allow approximation of the edges, or the use of sliding flaps, should be avoided, owing to the difficulty in controlling hæmorrhage from these incisions. If the wound is already infected, pack it open with gauze soaked in eu-flavine 1 in 2,000 and change as needed.

Wounds of the Skull, with or without Depressed Fragments, and without Penetration of the Dura.

After excision of the edges of the scalp wound and control of hæmorrhage, exposure is maintained by a self-retaining retractor of the large mastoid variety, and a thorough inspection is made. "Swish" the wound out well with normal saline solution. Remove all loose fragments of bone and all smaller depressed ones. Large depressed fragments may be elevated by an elevator placed under them through a burr hole, made with a Hudson's drill, in the undamaged skull about an inch away. Trephines are no longer used. Linear fractures must be carefully examined for retained hair and foreign bodies between their edges. If none is seen, and if there are no neurological signs of damage underneath, they may be left alone. The same advice applies to the opening or not of the dura. Interlocked depressed fragments of badly damaged bone may be removed either *en bloc* by placing burr holes around the area and connecting them with a Gigli's saw or with Montenovesi nibbling forceps, or else by knibbling them away with double-action nibblers of the Stille type. The older kind of rongeurs break the bone, and are very upsetting to a patient under local anæsthesia.

Fractures involving the base of the skull are always serious because of the risk of meningitis, especially if the ear is involved. The external meatus should be gently swabbed out till clean; in no circumstances should it be syringed; and a wick of gauze soaked in an antiseptic solution, such as 1 in 1,000 acriflavine, or in some aniline dye, should be lightly inserted and changed frequently. If the paranasal sinuses are involved, antiseptic nasal drops with argyrol *et cetera* should be used frequently. The immediate prophylactic use of sulphanilamide and sulphapyridine, one tablet of each every four hours, should be started. As well as "mastoids" and sponges, squares of lint will be found useful to cover over exposed areas of dura and bone edges during the operation. Hæmorrhage may thus be stopped in time, otherwise the use of Horsley's wax or a diathermy machine may be needed.

Wounds of the Dura and Venous Sinuses.

If the *dura mater* looks dark blue or yellowish, it should always be opened even if it is not lacerated, as a subdural hæmatoma may be underneath. This can then be allowed to escape, or washed away, together with any pulped brain substance; and if no fresh bleeding occurs, the opening can then be closed again to prevent a superficial infection from spreading deeply. If the superficial structures of the wound are already septic, it is better not to open an intact dura through the wound.

If the dura has already been lacerated by bone fragments or missiles, after they have been removed wash away old blood clots, damaged brain *et cetera* and close the gaps in the dura as far as possible after securing hæmostasis. If any of the larger venous sinuses of the dura are torn, the bleeding will have been profuse and will probably have proved fatal in the first few hours. If not, the repair of a tear in a venous sinus is very difficult, and is best accomplished by the application of a piece of hammered-out muscle graft, taken from the temporal muscle or elsewhere in the body, or from some other patient. The intracranial venous pressure should be lowered as much as possible before the graft is applied by raising the patient's head; and the graft should be kept in place for about ten minutes with digital pressure over cotton wool "patties" until it has stuck fast. If the sagittal sinus has been completely torn across each end must be tied off with a fine silk ligature, but the outlook for a return of cerebral function is then bad.

Wounds of the Brain.

Wounds of the brain are, of course, the most difficult to deal with if further damage is to be avoided and all the details of modern neuro-surgical technique must be employed. Gentle lavage from rubber ear syringes with Ringer's solution at body temperature is employed for *débridement* purposes. Cotton wool or "Lintene" "patties" are used for swabbing, and suction applied to them from a glass sucker keeps the field free of blood. Only the gentlest retraction with brain spatulæ over squares of "Lintene" is allowable, as pressure results in contusion and œdema afterwards. The careful sealing off of "bleeders" with diathermy coagulation, silver clips or fine ligatures is essential. The removal of all fragments of bone and all foreign bodies seen in the X ray film must be carefully checked. The neurological signs arising from the damage to the brain will not clear up at once, but only as the products of contusion are absorbed later, and if cerebritis and brain abscess are prevented. The lacerated edges of the dura are best not excised, as they rapidly become adherent to the underlying portion of the brain and prevent the spread of infection to the general meningeal spaces. They may be drawn together without tension as far as the swollen brain will permit. If a deep track in the brain exists, it should be cleared of foreign bodies and débris by catheter and suction from a glass syringe, or rubber bulb, as advocated by the late Harvey Cushing. Any accessible bone fragments or missiles which cannot be thus removed should be gently extracted by forceps similar to those used for removing stones from the bile ducts. Sometimes the wound track may reach the lateral ventricle. This makes the prognosis much more serious; but the toilet and removal of foreign bodies should still be completed. A Killian's type of nasal speculum is very useful for these procedures.

Foreign Bodies and Epilepsy.

Foreign bodies are usually pieces of shell; the deeper in they are, the smaller they are found to be, because if the larger ones penetrate deeply they usually kill. They must always be accurately located in stereoscopic X ray films. If they are situated more than an inch from the surface, no attempt should be made to remove them, unless an

effective electro-magnet is present, which can be applied at the end of a sterile metal bar passed down to the fragment. If forceps are used, permanent damage may be done to an important area or uncontrollable hæmorrhage may be started. It is not by any means certain that an abscess will form around foreign bodies, although, as a principle, they are better removed. If epilepsy develops later, it is just as likely to be due to the scarred track in the brain as to the foreign body; yet its removal is indicated, to give the sufferer the benefit of the doubt. Late epilepsy may be expected in a large number of cases in which the dura has been penetrated, especially if the cortical scar remains adherent to the scalp. It may not develop for fifteen to twenty years. Epileptic fits occurring in the early stages after the trauma are due to local hæmorrhage or œdema, together with contusion of the brain. They can be controlled by an intramuscular injection of "Dial" or "Nembutal", by the administration of "Luminal" and bromides by mouth, or finally, by a basal dose of "Avertin" *per rectum*.

Meningitis, Ventricular Infection and Hernia Cerebri.

Despite expectations, diffuse meningitis is a rather rare infection, if the wound has been adequately dealt with early enough, as the œdema in the surrounding contused brain obliterates any dead space and seals off the affected area. A mild degree of pleocytosis is frequently found in the cerebro-spinal fluid, only to subside later without the growth of organisms. More serious, however, are the cases in which a deeply spreading cerebritis reaches a lateral ventricle, so that the organisms are then rapidly washed through the remainder of the ventricular system and out via the foramina of Magendie and Luschka, to infect the subarachnoid spaces and leptomeninges diffusely. Once the diagnosis of a true meningitis is made on physical signs and by bacterial culture, the appropriate chemotherapy with sulphanilamide or sulphapyridine, or both, in full therapeutic doses, should be started at once; six to eight grammes should be given in the first twenty-four hours. Lumbar puncture should be repeated at intervals of eight hours, and the excess of fluid should be evacuated and submitted for a bacterial and cytological examination. On each occasion the pressure should be reduced to normal limits. Sometimes, when the patient is cooperative or very stuporose, continuous lumbar drainage can be maintained, whilst copious fluids are administered by all routes to promote lavage with cerebro-spinal fluid.

If the wound becomes infected, or if the edges of the closure of the scalp slough, the wound will gape and the damaged brain protrude to form a *hernia cerebri* or fungus. It should be carefully protected by a raised-up dressing built at some distance around it and by the frequent direct application of a hypertonic saline pack, to act as a dehydration measure. Further herniation, which will bring out a ventricular diverticulum and render the old method of shaving off the fungus unpardonable, can be kept in check by repeated or continuous lumbar drainage; and as hernia always indicates encephalitis or abscess, antiseptic drugs and serum must be persisted with. The importance of "pushing" the sulphonamides to tolerance cannot be over-stressed; and the value of sulphapyridine in cranio-facial injuries with resultant pneumococcal infections, so deadly in the Great War, should be immense. As the infection subsides the hernia becomes smaller and is covered over with granulation and eventually a thin epithelium. Excision and bone grafting to fill in the defect in the skull may be carried out later.

Post-Operative Treatment.

After careful suturing of the galea and skin, with soft tubes or corrugated rubber strips inserted at each end, the area is liberally painted over with Bonney's blue and covered with a dry dressing held securely and accurately in place by a starch or crape bandage, applied to grip below the supra-orbital ridges and the occiput. It is changed as often as is necessary to keep the pads dry. Administration of fluids intramuscularly may be needed every day for a few days; and, of course, accurate observations of the state of unconsciousness, of the pulse and respiration rates and of the temperature should be kept hourly till all the conditions are normal. Only thus can the signs of intracranial trouble be detected early enough to be of value. Finally, the use of a Lawson-Tait type of bed allows correct posturing and nursing to be more simply and comfortably carried out.

Acknowledgement.

The excellent articles on "War Wounds of the Head" by Geoffrey Jefferson, published in *The British Medical Journal* of August 12, 1939, and of August 19, 1939, have been largely drawn on for the compilation of this paper. The remaining procedures advocated are the result of experiences in the Neurosurgical Unit of the Royal Prince Alfred Hospital.

Notes on Books, Current Journals and New Appliances.

A BOOK OF SHORT STORIES.

"PHYSICIANS' FARE" is a book of attractive short stories which will be welcomed by medical readers.[1] The author, presumably a medical man, has a gift for narrative and for surprises. All the stories have a medical tang about them and they cover murder, mystery and the ordinary things of life. Not one is dull. "A Nice Quiet Locum-Tenency" is a startling tale of a young woman graduate who acts as *locum tenens* for a general paralytic; "Murder on the Fen" reveals the murderer by a point in anatomy; "Beloved Brethren" shows how some medical men in a small place love one another; and so on. These stories can be recommended as likely to appeal to anyone with medical knowledge; most of them are reprinted from magazines and hospital journals. It is a pity that the price is so high.

[1] "Physicians' Fare", by C. G. Learoyd; 1939. London: Edward Arnold and Company. Crown 8vo, pp. 302. Price: 7s. 6d. net.

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RADIOLOGICAL SURVEY OF THE SECOND AUSTRALIAN IMPERIAL FORCE.

In the interval which has elapsed since the war of 1914-1918, radiological surveys of large sections of the population, directed towards the detection of pulmonary tuberculosis, have been carried out in different countries. The many accredited reports recording the findings in routine radiological examinations of the apparently healthy now constitute an extensive literature, the burden of which is that very many active tuberculous pulmonary lesions are beyond detection by the most carefully and competently applied clinical methods. This statement needs no amplification and the disquieting fact has to be faced, with its corollary of the dissemination of infection by those least suspected and its bearing upon public health organization for the control and progressive reduction of tuberculosis. In the issue of this journal of January 13, 1940, we drew attention to the efficacy of the technique now elaborated for bacteriological investigation to supplement radiology in establishing the existence of subclinical pulmonary tuberculosis, and endeavoured to show that bacteriological endorsement of radiological findings could be adduced in a high proportion of instances.

The principle that no physical examination should be considered complete without a fluoroscopic inspection or radiogram of the thorax has been applied in many countries to such groups as students, nurses and industrial employees. Fluoroscopic examination has been utilized to discover indications for more intensive study by the radiographic film. Adopting such an approach, H. H. Fellows instituted radiological examinations of all employees and applicants for employment at the home office of the Metropolitan Life Insurance Company of New York.[1] In this way Fellows disclosed the presence of significant active pulmonary tuberculous lesions, subsequently substantiated, in 141 persons, the majority of whom were apparently well and exhibited no physical signs. The desirability of radiological surveys of large sections of the population with the object of seeking out symptomless but none the less infective cases of pulmonary tuberculosis has long been recognized, but hitherto the attendant expense has proved an insuperable barrier. The development of miniature radiography, however, has reduced to a minimum the expenditure involved, both in time and in money. This subject was discussed editorially in this journal on October 21, 1939; but readers may be reminded that the technique, in outline, consists in photographing the fluoroscopic image on 35-millimetre film in a camera of "Leica" or "Contax" type; the small photographs are examined by projecting them to a size approximating that of the standard film of chest radiography. By the use of miniature radiography the cost of a radiogram of the chest is reduced to less than twopence and films may be taken at the rate of more than one per minute. Following the war of 1914-1918 pulmonary tuberculosis among returned soldiers became a major problem in Australia, and one of great perplexity to medical boards and repatriation officers who were called upon to decide to what extent the disease was referable to war service and the contingent question of pension rights. From the facts established by mass radiological thoracic surveys in many countries in the intervening years it cannot be doubted that many men with active and arrested

[1] *The American Journal of the Medical Sciences*, October, 1934.

pulmonary tuberculosis were accepted for service overseas; that a large number failed to withstand the arduous conditions of active service is not only comprehensible, but is seen to have been inevitable.

The Australian Army Medical Corps Command, alive to the vitally important issues involved, has instituted systematized radiological examination of the entire personnel of the Second Australian Imperial Force. In this matter it received assistance from the Commonwealth X-Ray and Radium Laboratory of the Commonwealth Department of Health. For some time past research in miniature radiography has been carried out at this laboratory and its findings in regard to technique and apparatus were placed at the disposal of the Defence Department. It is required that all men in whom evidence of pulmonary tuberculosis is discerned by miniature radiography shall be reexamined by the standard thoracic radiogram. They are then considered individually by carefully constituted medical boards, for whom the results attending bacteriological examination are to be made available.

In this way the Australian Army Medical Corps administration is effectively carrying out a large scale measure of the first importance in promoting the physical efficiency of the fighting forces. Not only are men unfit for service eliminated, but soldiers with active and therefore infective lesions are removed from close contact with their fellows in camp hut and troopship. From the economic aspect the large scale radiology, now shorn of its formerly prohibitive cost, is not only justified but imperative if the very serious drain on the national expenditure occasioned by pulmonary tuberculosis in returned soldiers after the last war is to be obviated.

The radiological survey of the Second Australian Imperial Force, with its supplementary bacteriological investigations, is to be regarded also as an extremely important research. Facts will be established regarding the existence and number of tubercle bacillus "carriers" among a large and presumably healthy section of the population. Already a number of men, whose pulmonary disease was first detected by miniature radiography and who at the time of their enlistment were following their civil occupations in apparently normal health, have been shown to be actively discharging tubercle bacilli. Moreover, there is an obvious object lesson in the effective exploitation of miniature radiography by the army for the better control of pulmonary tuberculosis in the civil community. We have no doubt that the officers directing the anti-tuberculosis activities of the several States and the Commonwealth are fully seized of the importance of radiography and duly appreciate that measures such as the reporting of the tuberculous, their supervision in sanatoria, and the examination of known contacts leave much to be desired. The hitherto high cost has been an insurmountable obstacle to the drag-net radiological examinations which alone will detect pulmonary tuberculosis in its asymptomatic phases. Many persons thus affected spread infection unknowingly, and the means are now at hand for the unmasking of pulmonary tuberculosis at a stage most favourable for the successful treatment of patients and the control of the disease from the public health standpoint. We suggest that the subject is one to which life insurance companies and business organizations employing large staffs might direct their attention. Many will no doubt have noted the correspondence urgently advocating the routine X ray examination of militiamen sustained for many months in *The British Medical Journal*. That this correspondence has been so prolonged implies a reluctance on the part of the British authorities which we find difficult to understand. Our allies, the French, have practised routine radiological examination in the army for several years, and it has been adopted in Belgium, Switzerland and Germany. Few could fail to be impressed by such findings as were recorded by A. B. Robins[1] in his account of the study of applicants for employment in the Fire Department of the city of New York. This group embraced 1,964 candidates, young men in good health and of splendid physique, all of whom had passed strenuous physical tests and a rigorous medical examination. By routine chest radiography almost 2% of this carefully selected group were

[1] *American Review of Tuberculosis*, October, 1938.

shown to have had pulmonary tuberculosis, and in two-thirds of those so detected the disease was active.

The Australian Army Medical Corps Command is to be congratulated on the introduction and efficient operation of a measure which will do much to reduce invalidity in the forces now proceeding overseas; it is also designed to forestall enormous government expenditure in the care of invalided soldiers and the payment of pensions. On a future occasion we shall return to other aspects of this subject; but in the meantime it can be stated that the radiological survey of the Second Australian Imperial Force, supplemented by bacteriology, will also stand as a very valuable research from which data of great moment in Australian public health administration must emerge.

Current Comment.

MALE SEX HORMONES.

"Be mine the delight of maiden's caresses!" eagerly exclaimed Faust, palsied with age as he was, and at his interview with Mephistopheles he readily bartered his soul for youth and strength. Since the time of Dr. Faustus the quest for the elixir of youth, which is a euphemistic term for something to restore lost sexual capacity, has grown much keener. Philtres there are still, aphrodisiacs, vitality pills, tablets in handsome boxes purporting to contain extracts from the testicles of large and powerful animals, and those who buy them are bled. But during the last few years we have seen not only a rising tide of well-paid charlatanry but the fruition at last of a hundred and fifty years' scientific work directed first to the physiological demonstration and later to the isolation of an internal secretion of the testis.

The attempts of John Hunter at gonadal transplantation in fowls, well known to those who have visited the museum of the Royal College of Surgeons in Lincoln's Inn Fields, were made in the eighteenth century and were followed up in the nineteenth by various workers, notably Bertholdt; but testicular transplantation in man was not seriously attempted until 1912. Still, despite the introduction (and exploitation) of "monkey glands" and the wider understanding of the tonic effects of the operation of vasotomy, which still enjoys a good vogue among urologists, the internal secretion of the testis remained intangible until 1927. In that year U. C. McGee was able to extract from the lipoid fraction of human male urine an androgenic substance, that is to say, a substance capable of repairing the atrophy of accessory sexual organs in castrated animals. During the next few years a number of workers obtained a similar substance from human urine and from bulls' blood; but no notable advance was made until in 1931 A. Butenandt, who had extracted with chloroform over three thousand gallons of human male urine, obtained the androgenic substance in pure crystalline form. In 1933 he suggested its chemical structure and called it "androsterone". Soon after Butenandt suggested its structure L. Ruzicka and his collaborators, by a brilliant piece of work, prepared it from cholesterol by means of a process of chemical degradation; their paper was published in 1934. In 1935 K. David, E. Laqueur and their fellow workers extracted a pure crystalline substance of much greater androgenic potency than androsterone from bulls' testes and called it "testosterone". In 1935 also Ruzicka and A. Wettstein, and almost simultaneously Butenandt and G. Hanisch, suspected by analogy with androsterone that reduction of a certain keto group in the molecule of androstenendione (a less potent oxidation product of androsterone) would yield a compound of enhanced activity; this not only proved to be the case, but the workers found that they had synthesized testosterone. Its production on a commercial scale followed at once.

In 1937 R. Deansley and A. S. Parkes reported the extraordinary effectiveness of pellets of crystalline hormones implanted beneath the skin of birds, and in the same year J. E. Howard and S. A. Vest began experiments of placing pellets of several milligrammes of testosterone in the subcutaneous tissue of some of their patients, eunuchs in whom they had already restored *libido* and sexual potency with injections of the same substance contained in a vehicle of sesame oil. The experiments were a failure; but later they used much larger pellets, containing 100 to 800 milligrammes of testosterone, and have recently reported the results, which have been very gratifying.[1] In 1938 it was reported by G. L. Foss that testosterone exhibits its androgenic properties in the human subject when incorporated in an ointment and rubbed into the skin. The large number of papers which are now appearing in medical journals all over the world indicate that the place of testosterone in the treatment of testicular deprivation and eunuchoidism is well established, while numerous experiments have been made and are being made, some well controlled and others savouring of commercialism, on the use of testosterone for such disorders as enlargement of the prostate, uterine bleeding due to unopposed œstrogenic action, the induction of temporary sterility in women (for it has been found that testosterone inhibits the progestational stage of the development of the endometrium), chronic mastodynia, as well as for senility and such vague conditions as "a feeling of diminished virility". Overdose of the hormone is attended by such symptoms as excessive sexual activity and painful priapism in the male

[1] *The American Journal of the Medical Sciences*, December, 1939.

and hirsuties and enlargement of the clitoris in the female.

What of the future of the therapeutics of androgenic substances? The number of persons unfortunate enough to need treatment on account of castration or eunuchoidism must be very small, and eunuchoidism is often diagnosed in error. Doubtless other limited fields for the therapeutic use of male sex hormones will be developed along the lines that have been indicated. But a million Fausts still seek the elixir of youth; and in a sadder plight than these there is a great mass of people secretly suffering mental anguish on account of fancied sexual weakness due to proneness to masturbation, poor physique, precocious ejaculation, nocturnal emissions, organic nervous disease, mild depressive psychosis, chronic fatigue, valetudinarianism and the like; and they will be ready to spend a fortune on anyone who advertises in the newspaper that he will give them "strength" or "vitality". There is danger in the uncontrolled use of these androgenic medicaments. Already we have seen the marketing of complexion creams containing œstrogenic substances. Before the charlatan and the patent medicine vendor add hormone therapy to their other weapons, the sale of these agents containing hormones should be controlled by law.

IS BREAD WORTH WINNING?

A FEW years ago it was a matter of individual taste and preference whether one ate wholemeal or white bread. But recent advances in knowledge of vitamin B_1 requirements now place the eater of white bread amongst the sufferers from nutritional deficiencies. It is now scientifically realized that it is impossible to construct a nutritionally satisfactory diet using white bread as the main source of cereal. So serious is this situation that Dr. G. R. Cowgill,[1] in a report to the Council on Foods of the American Medical Association, discusses the possibility of adding synthetic vitamin B_1 to all cereal foods as one practical and rational means of solving the problem. According to the report, adults require as a minimum 300 to 350 international units of the vitamin. Stiebeling and Phipard set the requirement at 500 international units daily. Children require approximately two and a half times as much vitamin in proportion to the total Calorie intake. The point at issue is, then, to what extent are these requirements likely to be met in the present average dietary.

It has been realized for some time that the dietaries in common use in the Orient and other parts of the world where beriberi is endemic are seriously deficient in vitamin B_1; but until quite recently it was thought unlikely that this should be true also for the diets of Western civilization. However, the recent increased therapeutic use of pure vitamin B_1 (thiamin), made possible by its chemical isolation and synthesis, has revealed numerous and remarkable successes in treatment with this factor, often in conditions in which deficiency seemed quite unlikely. And it appears that vitamin B_1 deficiency may be more widespread in civilized countries than has been recognized previously. An analysis of statistics of food consumption lends further support to the contention. Cowgill has estimated that the average American secures probably as much as 25% of his Calories from wheat and its many products. Most of this, of course, is consumed as highly refined material, the product of modern milling processes, which, it is pointed out, has a vitamin B_1 content approximately one-eleventh of that of the old stone-ground flour. It is generally agreed that the intake of vitamin B_1 among Western peoples is only about one-fifth of what it was sixty years ago. If we consider also the amount of sugar used, we find that the American people consume something like 42% of their Calories in the form of these two foodstuffs, relatively poor in vitamin B_1 or free of it—a point made by Gutteridge with reference to the Australian diet in an article published in this journal on February 1, 1936. Moreover, these figures have been reached with the use of 3,000 Calories per day as the base of reference. If smaller figures, 2,700 or 2,500 for example, are used, the percentages become 55 and 60 respectively. The vitamin B_1 content of this enormous fraction has been estimated to be only about 50 international units, that is, roughly one-tenth of the daily requirement. Thus, for nutritional adequacy, 90% of the vitamin B_1 requirement must be supplied from the remainder of the dietary. However, foods such as milk, green vegetables and fruits, while excellent sources of calcium and vitamin C, do not rate high in vitamin B_1. Egg yolk and meat (especially pork) are more reliable sources; but if 50% to 60% of Calories in a diet are derived from white flour and sugar, eggs are not likely to be included in sufficient quantity to be a significant source of supply, and the actual value of various meats as a source of vitamin B_1 is still uncertain. In the light of recent knowledge, the tendency among research workers is to recognize cereals as the most suitable carrier of vitamin B_1 in the average dietary. The problem then becomes one of increasing the vitamin B_1 content of the cereals as consumed.

Two constructive lines of action present themselves. First, a more intensive plan of public education might be instituted concerning the relative nutritive values of white and wholemeal or wheat germ cereal products. This method will necessarily be slow and, if past experience be any indication, only partially satisfactory. The development of modern transport facilities, the necessity of marketing cereals in forms that are stable for long periods, and the prevailing preference for white bread (by both consumer and producer) will probably continue to give white flour a position of prominence. Moreover, with the small change-over

[1] *The Journal of the American Medical Association*, December 9, 1939.

to wholemeal which has already occurred, some milling companies declare that they have difficulty in meeting supplies of bran and pollard. Bakers and millers show a definite resistance to change from white flour to nutritionally superior foods. A second line of attack is suggested by the chemical synthesis of pure vitamin B_1. Cowgill states that it is possible to add the vitamin to wheat flour in any desired and appropriate amount; and it seems reasonable to believe that the one act of adding the vitamin to wheat flour (before transformation into its many products) would increase directly and satisfactorily the vitamin B_1 intake of the great majority of our people. The suggestion has unlimited possibilities, and a host of secondary questions immediately arise. How much thiamin should be added? Should sufficient be added merely to restore the B_1 content of the flour to that of the original unmilled cereal? Or should the flour be fortified with greater amounts of the vitamin? If so, then to what extent? And is the cost of such addition within the economic limitations of the "bread-winner" (with the doubt whether the white bread is worth winning)?

Another milepost has been reached along the road of nutritional progress. While synthetic substances have been used in curative therapeutics, this is the first serious suggestion that a synthetic food be used in what might be termed constructive therapeutics.

DRAINAGE OF THE PERITONEAL CAVITY.

Drainage of the peritoneal cavity has remained a problem since surgeons first invaded its precincts; this problem is today perhaps regarded as of greater complexity than it was by the pioneers of abdominal surgery. For many years the comfortable doctrine of Lawson Tait prevailed: "When in doubt, drain." Objections to this simple plan gradually began to be raised, some on experimental, some on clinical grounds. St. Leger Brockman took exception, partly at least, to the very simplicity of the plan, when in 1924 he wrote in *The British Journal of Surgery*: "Admirable as such advice has been and is, we must remember that doubt spells ignorance, and complacently to submit one's actions to the ruling of such a motto is to spend a life of surgical stagnation." The question of drainage of the peritoneal cavity arises most frequently in the treatment of peritoneal infection associated with acute appendicitis, and discussion has been based on the clinical observation of ill effects attributed on the one hand to the presence of a drain and on the other to failure to use a drain. Thus Trinca has maintained in this journal (October 7, 1933) that drainage is employed too frequently and without sufficient thought. By contrast, Newton, writing in *The Australian and New Zealand Journal of Surgery* of January, 1939, supports the old dictum of Lawson Tait, and sees cause for alarm in the growing advocacy of restricted use of drainage. The results of experimental work on drainage of the peritoneal cavity are of great interest, and it is possible that not enough attention has been paid them by clinical observers. Investigations, such as those of Yates in 1904, throw grave doubts on the feasibility of securing effective drainage by the methods in general use. This worker inserted a drainage tube through the abdominal wall in a series of dogs and found that after a period averaging about six hours communication between the tube and the peritoneal cavity was shut off, and a solution of dye introduced even under pressure into the tube could not be made to reach the cavity or *vice versa*. Other workers, using suspensions of organisms in place of dye solutions, have shown that the peritoneum cannot be infected from a drainage tube after about twenty-four to forty-eight hours. These results suggest that one factor in drainage has been neglected, and that is the time factor. If after a limited period of from six to forty-eight hours a drainage tube ceases to communicate with the general peritoneal cavity, then it becomes clear that efforts must be directed towards the securing of the maximum drainage in the period available. Probably the best way of achieving this is to make use of gravity. Harbin in 1905 reported the treatment in the prone position of a patient with diffuse purulent peritonitis, and wrote:

> During the first six hours the drainage was very large in amount, saturating the dressings and sheets. The amount of discharge in this case by far exceeded what I have ever seen from any other abdominal wound, and the amount of toxic material in the peritoneal cavity thus eliminated explained the rapid improvement of his symptoms and final recovery.

E. G. Williamson and L. M. Rankin,[1] after making use of the preceding quotation, refer to reports by other workers who have employed the ventral decubitus to assist peritoneal drainage with satisfactory results. Williamson and Rankin treated by use of the prone position thirty-two patients who were suffering from general peritonitis following acute appendicitis with perforation, and none of them died. The general plan was as follows. The peritoneum was closed around one to three Penrose rubber-covered cigarette drains, the remaining layers of the abdominal wall were left unsutured and the wound was packed with gauze. When the patient returned to his bed he was immediately placed on his abdomen, the head of the bed being elevated eight to twelve inches. In each instance the dressings rapidly became saturated, necessitating frequent changes during the first twenty-four hours. The amount of discharge is described as far exceeding that seen when the patient is in the Fowler or other position. After thirty-six to forty-eight hours the patients were turned onto their backs. As stated before, there were no deaths, and it is claimed that the ultimate results were excellent. The present position of the treatment of general peritonitis is unsatisfactory, and it would seem that where applicable this method of improving drainage should have an extended trial.

[1] *The American Journal of Surgery*, September, 1939.

Abstracts from Current Medical Literature.

PATHOLOGY.

The Histology of Regional Ileitis.

G. HADFIELD (*The Lancet*, October 7, 1939) states that the earliest and possibly the specific pathological process in regional ileitis is lymph-adenoid hyperplasia with the formation of non-caseating giant-cell systems in the submucosa. This process is also present in the regional lymph glands, even though ulceration may have obscured the nature of the primary lesion in the submucosa. Acid-fast bacilli cannot be demonstrated histologically in the lesions. From the purely histological point of view the appearance, evolution and retrogression of the giant-cell systems of regional ileitis resemble the tissue reaction of sarcoidosis rather than that of tuberculosis.

Further Observations on Paget's Disease of the Nipple.

R. MUIR (*The Journal of Pathology and Bacteriology*, September, 1939) reports on the examination of 42 cases of Paget's disease of the nipple. His examination supports the view that the nipple lesion is due to intra-epithelial growth of cancer cells which have spread from malignant disease in the upper extremity of a lactiferous duct—intraduct carcinoma. It also supports the view formerly expressed by him as to the association of Paget's disease with carcinoma of the mamma. In all cases intraduct carcinoma was present in the nipple. Of 39 cases fully examined, it was present in both breast and nipple in 34; in 30 it was accompanied by infiltrating carcinoma of the breast. The carcinoma of the breast which is so often associated with Paget's disease is ordinarily due to extension from an independent focus of intraduct carcinoma of the breast. In view, however, of the mode of spread of intraduct carcinoma, the possibility of carcinoma of the breast resulting from a downward direct extension of intraduct carcinoma in the nipple must be admitted; but no undoubted example of this has been met with in the author's series of cases. On the other hand, in six cases of carcinoma of the breast there was found an upward spread of intraduct carcinoma from a level below the nipple, and this had just reached or was just reaching the epidermis before the appearance of the clinical signs of Paget's disease. The time of appearance or the stage of the nipple lesion gives no indication whatever as to the condition of the breast in Paget's disease. The Paget lesion may mean that the first seat of malignancy is in a duct in the nipple, or it may be a terminal phenomenon when there is already extensive malignant disease in the breast. The cases recorded show that whatever the stage of the nipple lesion, in the great majority of cases intraduct carcinoma is present in the breast and that in a large proportion of these ordinary carcinoma has developed. Anything in the nature of local treatment of the disease of the nipple is accordingly quite unjustifiable.

Observations on the Musculature of the Lung in Children.

S. ENGEL AND G. NEWNS (*The Journal of Pathology and Bacteriology*, September, 1939) refer to a paper of Baltisberger, published in 1921, which is practically the only one that has dealt extensively with the musculature of the lung. Although Baltisberger's paper is based on the examination of a single lung, his conclusions have been generally accepted since they were published. He had described the presence of a muscular network surrounding the respiratory bronchioles and the alveolar ducts, the presence of numerous bundles of smooth muscle in the interstitial connective tissue separating the individual lobules from each other, and a one or two layer muscular coat within the pleura. During another line of investigation these results became questionable to the authors, and therefore they examined especially from this point of view the lungs of 196 children within the ages of six weeks and twelve years. The result of the examination is that there is no muscle at all in the pleura or in the interstitial tissue of the lung, and only very fine, but not thick, muscle bundles surrounding the air spaces. They have in their series one case in which muscular tissue was found in the same arrangement as described by Baltisberger. As they have seen under pathological conditions only hypertrophy of the muscle fibres surrounding the air spaces, but no musculature in the interstitial tissue and the pleura, they suggest that in this case of theirs, as well as in the case of Baltisberger, a congenital abnormality was present. Muscular tissue in a normal lung therefore occurs only within the structures of the bronchial tree, but not in the interstitial tissue or in the pleura.

Fibrosarcoma following the Intra-articular Injection of Radium Chloride.

FLEMMING NORGAARD (*The American Journal of Cancer*, November, 1939) reports on the case of a woman who was admitted to hospital at the age of fifty-six years for a tumour in the right knee joint. She had had joint symptoms from the age of eleven years, beginning in the left wrist and elbow. Symptoms occurred in the right knee joint at the age of thirty, and at the age of forty-four in the right shoulder. The patient had tried many forms of treatment. In 1928 she went for the first time to St. Joachimstal, in Bohemia, where she was given treatment at the radium springs with excellent results. Later, the symptoms recurred and she again received treatment at St. Joachimstal in 1929, 1931, 1934, 1937, and declared after the last visit that she felt better than she had ever felt before. Pain recurred shortly afterwards in the right knee joint, and although objectively there was no change from the former condition, the symptoms became worse in spite of any treatment. After a lengthy stay in hospital the final diagnosis, fibrosarcoma of the epiphysis of the tibia, was made and verified by microscopic examination after amputation of the leg. Inquiries at St. Joachimstal revealed that the treatment there had consisted not only of the external application of radio-active substances, but that 10 microgrammes of radium chloride had been injected into the knee joint. Tests for radio-activity carried out on the tumour tissue and parts of the femur and tibia adjoining the knee joint gave positive results. Examination of the bone marrow of the tibia and femur showed evidence of radium damage. The author concludes his paper with a stern warning against the administration, whether by oral or parenteral routes, of even minimal quantities of radioactive substances to the organism. Such administration ought never to take place either for therapeutic or diagnostic purposes.

Formation of Coronary Thrombi.

J. C. PATERSON (*The Journal of the American Medical Association*, March 11, 1939) reports the history and pathological examination of two cases of fatal coronary occlusion. It is suggested that high coronary blood pressure, the result of strenuous exercise or emotion, is one of the underlying causes of capillary rupture and intimal hæmorrhage of arteriosclerotic coronary arteries. If conditions are favourable, the various changes resulting from capillary rupture may then be the initiating factors in the deposition of coronary thrombi. The formation of these thrombi is a gradual process, sometimes occupying several days before occlusion of the coronary lumen, with its resulting cardiac pain, is produced. Therefore the activities of a patient immediately preceding the onset of an attack of coronary thrombosis have no relation to the ætiology of the precipitation of a thrombus, but are purely coincidental. The pathological appearances in a series of fatal cases of coronary thrombosis suggest strongly that excessive exercise and emotional stress are intimately concerned in the mechanism of coronary artery thrombosis.

An Ovarian Teratoma Containing Cerebellar Tissue.

R. A. WILLIS (*The Journal of Pathology and Bacteriology*, November, 1939) describes an ovarian teratoma

which presents a number of very unusual structural features. The main cyst is not a dermoid cyst lined by skin, but an enteroid cyst lined by intestinal epithelium. The specimen therefore emphasizes the inaccuracy of the usual name dermoid cyst as applied to benign ovarian teratomata. Paradental residues were found in association with teratomatous teeth. The most unusual feature, however, is that within the nervous tissue which was an abundant component of the teratoma, not only a large number of nerve cells and oligodendrocytes but an area of well-formed cerebellar cortex was present. It is the first finding of this kind in the author's series of over 40 carefully examined teratomata, and he was able to find the recording of only one other similar case in the literature.

MORPHOLOGY.

The Pigments and Colour of Living Human Skin.

E. A. EDWARDS AND S. Q. DUNTLEY (*The American Journal of Anatomy*, July, 1939) have investigated the colour of the skin in a number of living and dead human subjects. Use of the Hardy photo-electric recording spectro-photometer eliminated any subjective factor in colour estimations. The authors point out that light passes right through the skin and down to the subcutaneous fat, and that every layer through which the light passes introduces some optical modification of the resultant effect; in addition, the pigments melanin, melanoid, oxyhæmoglobin, reduced hæmoglobin and carotene, all contribute to the final result. In fair-skinned people melanin is sparse and is restricted to the basal cells of the epidermis; it increases progressively in brunette whites, yellow-skinned races and Negroes. Some melanin occurs in the melanophores of the dermis, but is negligible except in the eyelids and axillæ of certain individuals, and in the sacral patch of children of the yellow races in particular. Melanin is responsible for the yellow, brown or black appearance of the skin and possibly for the bluish tinge in the feathers of some birds. In the bluish shaven cheek melanin is present in the diagonally placed hair bulbs and shafts. Melanoid is probably a degeneration product of melanin, and it produces a purer yellow colour in the skin than melanin does. Oxyhæmoglobin exerts its maximum effect where the superficial blood supply is richest and fastest; reduced hæmoglobin where it is slower and more venous. The turbidity of the cells of the overlying *stratum mucosum* of the epidermis reduces the chromatic effect of oxyhæmoglobin; on the other hand, the blue colour of the larger superficial veins occurs when there is sufficient hæmoglobin to absorb all the incident light. Carotene is present in the subcutaneous fat, but is especially concentrated in the *stratum corneum* of the epidermis, where it is probably held by the lipins in the cells, particularly where the *stratum corneum* is very thick. The carotene is said to reach this superficial locus by intercellular infiltration from the dermal capillaries. Carotene is particularly abundant in females, being concentrated in the skin of the breasts, abdomen and buttocks. The generally lighter colour of the female depends upon the lower skin content of blood and melanin; on the other hand, the nape of the neck, the *linea nigra* and the axillæ show a higher melanin ratio than in the male. Racial variation in colour depends entirely upon variation in the melanin and melanoid content of the skin; there are no pigments other than those which normally occur in the white race.

The Innervation of the Interstitial Cells of the Testis.

H. OKKELS AND K. SAND (*Bulletin d'Histologie Appliquée*, November, 1939) describe a special nerve supply to the interstitial cells of the testis. The nerves concerned may be either myelinated or not, and they accompany the arteries distributed from the body of Highmore. These arteries either run in the interlobular septa or they pass peripherally under the *tunica albuginea*. The nerves which accompany the interlobular arteries are few and small, and they present collections of interstitial cells among their fibres. These intraneural collections are from 0·5 to 1·5 millimetres in length, and the cells present all the characters of the cells of Leydig, except that they are generally larger: 30μ to 40μ as opposed to the usual 15μ to 20μ. The relation of the peripheral nerve fibres to the cells is one of two kinds: either the cells form intraneural collections as above or the fibres end amongst groups of interstitial cells. The authors describe their techniques.

Recto-Uterine Venous Anastomoses.

E. G. WERMOUTH (*The Journal of Anatomy*, October, 1939) describes anastomotic channels between the rectal and uterine veins, that is, between the portal and systemic systems. He shows that blood does not flow from the uterine to the rectal veins under normal conditions, but considers that it may do so under pathological conditions and so spread embolic infection from the uterus to, for example, the liver. On the other hand portal obstruction may be relieved through these anastomoses, and the author believes that they are more efficient for this purpose than are the circum-anal anastomoses.

The Motor Cortex in the Phalanger.

F. GOLDBY (*The Journal of Anatomy*, October, 1939) presents an experimental investigation of the excitable cortex and various descending tracts in the Australian phalanger (possum). The excitable cortex is distributed before and behind the orbital sulcus, and electrical stimulation produces movements of the facial muscles, jaws, neck, tongue, forelimb, abdominal muscles and the hindlimb; there were no tail movements. The excitable cortex presents a well-defined fourth lamina. The pyramidal tracts decussate to the posterior columns of the spinal cord, and pyramidal fibres could be followed to the mid-thoracic region. Fronto-pontine fibres are few and largely non-myelinated; temporo-pontine fibres constitute at least the lateral one-third of the *basis pedunculi*.

The Soft Palate.

F. WOOD JONES (*The Journal of Anatomy*, January, 1940) has conducted an extensive comparative study of the soft palate. He shows that this is widely represented, occurring even in birds; on the other hand, the velum of crocodiles is not homologous with that of mammals. Originally a complete partition extending from the roof of the mouth to the dorsal wall of the pharynx, the velum becomes perforated to establish continuity between the nasal passages and the *rima glottidis*. The perforation should be called the *hiatus nasopharyngeus*, not the "pharyngeal isthmus", because the region above the perforation belongs to the nasal cavities, not to the pharynx. The palato-pharyngeus muscle in man comprises two portions: that which is usually called palato-pharyngeus is really the palato-cricoideus, while the true palato-pharyngeus is more or less included with the superior constrictor of the pharynx. The true palato-pharyngeus is really a constrictor of the *hiatus nasopharyngeus*. In many animals the *rima glottidis* extends through the *hiatus nasopharyngeus*; in others it is frequently or permanently withdrawn to lie at a lower level. In this case the glandular tissue in the anterior margin of the hiatus becomes aggregated in a mass which ultimately forms the uvula. In phonation the *rima glottidis* must be withdrawn from the hiatus if it lies there, and in animals in which phonation is a more or less constant phenomenon, as, for example, the apes and especially man, the rima is permanently withdrawn.

Hippocampus and Corpus Callosum.

F. GOLDBY (*The Journal of Anatomy*, January, 1940) shows that in the bat, mouse, rat and cat the hippocampus turns back under the posterior end of the *corpus callosum*. This, he considers, supports the view of Elliot Smith that the *corpus callosum* develops in the hippocampal commissure ventral to the hippocampus and is opposed to the view of Abbie that the *corpus callosum* has grown through the subiculum dorsal to the hippocampus.

British Medical Association News.

SCIENTIFIC.

A MEETING of the Western Australian Branch of the British Medical Association was held on September 10, 1939, at the Government Hospital, Kalgoorlie, DR. N. M. CUTHBERT, the President, in the chair. The morning session was devoted to a number of clinical demonstrations; in the afternoon and evening several papers were read. Part of this report appeared in the issue of March 23, 1940.

Summer Diarrhœa in Children.

DR. R. H. CRISP discussed summer diarrhœa in children. He said that gastro-enteritis was not a diagnosis in the full sense of the word. The diagnosis should include a consideration of the child's general constitutional type or diathesis, prematurity, exudative or seborrhœic indications *et cetera*. The feeding history of the child prior to the illness should be investigated and the state of nutrition should be determined—whether the child was dystrophic or overfed.

A detailed examination should be made, and should include a search for evidence of parenteral infection. Such infections were frequent causes of diarrhœa, particularly infections of the upper portion of the respiratory tract, the throat and ears and the urinary tract. The presence or absence of these infections should be noted at a routine examination, as they frequently occurred as complications in the course of the enteritis. The abdomen should be examined for the exclusion of intussusception whenever blood and slime were present in the stools. An enlarged liver suggested dysenteric infection.

Dr. Crisp then went on to discuss the clinical condition as an indication of the degree of toxæmia, acidosis and dehydration. He said that the first sign to be looked for was fever. Cerebral signs should then be sought; the state of consciousness should be estimated, and meningism and twitching or convulsions should be noted if present. The facies was the next thing to observe; enteritis implied an anxious look, and cherry-red lips, accompanied by air hunger and sweet breath, implied acute acidosis. The medical attendant should then note the skin turgor, the condition of the heart and peripheral circulation and the condition of the abdomen (for distension, enlargement of the liver or the presence of tumours); he should observe the condition of the fontanelle. The mouth, throat and ears should be examined, and the urine and stools should be inspected.

Discussing treatment, Dr. Crisp said that the first phase included a preliminary period of starvation and observation. The bowel should be emptied by a wash-out, which need not always be repeated. If the child was seen early in the illness, an initial purge was used. Dr. Crisp did not use castor oil or calomel, as these substances tended to make the patient vomit. He preferred milk of magnesia, the dose possibly being repeated, or a few doses of equal parts of sodium sulphate and magnesium sulphate; the dosage was ten grains of each given every two hours until six doses had been taken. If vomiting was occurring Dr. Crisp gave a mixture of magnesium carbonate, sodium bicarbonate and bismuth carbonate. To combat hyperthermia the child was bathed or sponged, and occasionally cool packs were used. A cool or air-conditioned room was a great boon, and saved lives during a heat wave. As a stimulant, to maintain the peripheral circulation, the mustard bath was invaluable. Brandy, "Cardiazol", caffeine, adrenaline and "Veritol" were other stimulants that were used. If twitching or convulsions were present, one-third of an ampoule (one grain) of "Luminal" was injected. If parenteral infections were present they were treated on routine lines. Children suffering from enteritis were infective, and should be isolated and treated as "contagious".

Dr. Crisp went on to say that the next problem was to correct toxæmia, dehydration and acidosis, and to replace fluids and minerals and maintain the fluid and mineral balance. Sugary solutions and lactic acid preparations inhibited the growth of dysenteric organisms in the bowel. Dr. Crisp then instanced several solutions that might be given freely and frequently; they were: boiled water with fruit juice and glucose, N/2 saline solution with glucose, weak tea, raisin tea and apple juice. Older infants were given water cereals, and if the apple diet was being used, apple tea or apple *purée*. The infusion of saline solution should not be long delayed in cases of toxæmia and dehydration; it was best not to wait for dehydration to occur. The fluid intake should be measured, and if it was inadequate, infusion should be proceeded with. Fluids could be given by the subcutaneous, the intraperitoneal or the intravenous route. Infusion by the continuous intravenous drip method (venoclysis) should be used in suitable cases; it might be a life-saving measure. The "Soluvac" fluids were convenient and satisfactory. Dr. Crisp said that he preferred Ringer's solution, or better, half-strength Ringer's solution, with 5% glucose solution. A suitable isotonic solution was six parts of normal saline solution with four parts of a 10% glucose solution. A convenient needle for intravenous use could be improvised by breaking off a small hyopdermic needle and leaving a butt of one-half to three-quarters of an inch; this was fitted into a short length of ureteric catheter. Five to eight ounces of solution were run in fairly rapidly in the first twenty minutes, and then the flow was regulated to about one ounce per hour until the toxæmia was corrected, in from thirty-six to seventy-two hours. Nothing was given by mouth for twelve hours; after that time sips of fluid were administered. Dr. Crisp stressed the dangers of hydræmia and chloride retention. He said that chloride retention was possibly prevented by the use of N/2 saline or Ringer's solution in preference to those solutions in normal strength. Caffeine, adrenaline or citrated blood could be injected into the tube below a clamp; citrated blood should not be injected until dehydration had been corrected.

Dr. Crisp then referred to toxic vomiting. He said that it was an acutely dangerous complication. A stomach wash-out with sodium bicarbonate solution was of value, and sodium bicarbonate or sodium lactate might be added to the drinks. Whey also was useful. A bismuth alkaline mixture might succeed in controlling the vomiting, and an injection of "Luminal" had been found very helpful. Dr. Crisp said that these were cases in which venoclysis should be considered. He then described another procedure which he had found to be life-saving. A fine catheter, graduated in ten centimetres, was passed orally into the stomach 20 to 25 centimetres from the gum margin. Peristalsis carried the tip into the duodenum 25 to 30 centimetres in twenty or thirty minutes, or even in a shorter time. The position was checked by injection and aspiration with a "Record" syringe. Large quantities of fluid could then be given without causing vomiting, and the liver was not excluded from early participation.

Speaking of acidosis, Dr. Crisp said that it might be clinically suggested by pallor and cherry-red lips, and by hyperpnœa with sweet breath. Infusions were called for; Hartmann's solution should be used (Ringer's solution and sodium lactate). After a stomach wash-out sodium bicarbonate with glucose or sodium lactate solution might be given by mouth. The last mentioned could also be injected intramuscularly or subcutaneously.

Dr. Crisp then went on to speak of abdominal distension. He said that it might be troublesome, and was combated by stomach or bowel wash-outs. A rectal nozzle and a tight binder, as well as an injection of pitressin or pituitrin, might help to get rid of much wind. Pineapple juice was a valuable remedy in abdominal distension from any cause.

Dr. Crisp then said that hydræmia might occur, especially in the presence of an exudative diathesis, independently of infusions. The tissues became soggy and the peripheral circulation poor. The administration of

sodium chloride was discontinued and the fluid intake was limited to the fluid needs (one-sixth to one-fifth of body weight). The peripheral circulation and the kidneys were stimulated by mustard baths and packs, plasters over the renal area and injections of adrenaline, "Veritol", "Cardiazol" *et cetera*.

The apple diet was then discussed by Dr. Crisp. He said that it represented a great advance in treatment. Raw scraped apple *purée* was given, as much as possible being administered every four hours. Extra fluids were given, ample to cover the fluid need. Dr. Crisp said that he found the "Granny Smith" the best apple to use. For infants aged under about ten months apple tea was used; this consisted of two parts each of scraped apple and normal saline solution and five parts of weak tea. Apple was not always well taken, particularly if vomiting was present. Sugar or glucose or saline solution or other fruit juices might be used with apple in an endeavour to administer large quantities. Bulky apple stools should begin to appear in about eight hours. Apple alone was given for twenty-four to forty-eight hours, and other foods were added as the quantity of apple was gradually reduced. If the child was old enough cereal jellies could be added, as well as broth, gruels, rusks, toast or biscuit with honey *et cetera*, mashed banana and potato and gravy. These could be added before the administration of milk was resumed, and then vegetable *purée* and white meat could be given. Dr. Crisp uttered a warning against the early resumption of milk, particularly milk fats. Rich, fatty milk foods like "Vi-Lactogen" *et cetera* were quite unsuitable, and a frequent cause of disaster. "Intermediate" milk preparations, which could be used as "stepping stones", were malted milk, Nestlé's condensed milk, buttermilk ("Eledon") or skimmed acid milk, or later, skimmed "Bengerized" milk mixtures. It was impossible to avoid milk foods for younger infants. Whey, buttermilk ("Eledon"), Nestlé's condensed milk and malted milk were used at first, and later skimmed acid milk or skimmed "Bengerized" milk mixtures. Cereal jellies and broth were given early if the child was old enough. Prolonged starvation was to be avoided after the acute stage was under control, especially if the disorder was of the lower bowel type (colitis) without toxæmia, vomiting, abdominal distension *et cetera*. Prolonged starvation led to loss of resistance and to food intolerance, so that relapses and feeding upsets occurred when increased feeding was attempted. During the convalescent period and for some time afterwards milk fats, oils, emulsions and "roughage" should be avoided. A dysenteric infection of the bowel wall took probably three weeks at least to heal, perhaps much longer. The child might be better before the bowel was, so there was a period when feeding liberties were likely to lead to relapses.

Dr. Crisp said that drug treatment was of secondary importance, and was symptomatic. Calcium carbonate and bismuth with chalk were bowel sedatives. Calcium carbonate was given especially if scalding was present; bismuth with chalk could be given with *Hydrargyrum cum Creta* in the late stage when the stools were offensive. Repeated small doses of *Oleum Ricini* were the traditional remedy in the late "irritable" stage; the same applied to "Tannalbin" and tincture of opium. The effect of tincture of opium had to be carefully watched. In the early stage, while elevation of temperature, toxæmia or offensive stools were present, no attempt should be made to "bottle up" the diarrhœa. Dr. Crisp said that he had not had good results from the use of bowel antiseptics such as salol, "Dimol", *Hydrargyrum cum Creta et cetera*; on the contrary, he did not use them, because they might be harmful, irritative and toxic. The sulphanilamide drugs did not favourably influence the course of the disease, but were used in septic complications such as otitis, pyelitis *et cetera*. Dr. Crisp had found the use of dysenteric antiserum disappointing; if it was to be used, it should be given early and in adequate dosage (30 cubic centimetres). Dr. Crisp said that he had not had enough experience in bacteriophage treatment to express an opinion as to its value.

Dr. Crisp went on to say that the cause of death in enteritis in the early stages was usually severe toxæmia and dehydration. During the course of the disease complications, often mild in themselves, might upset the balance and cause a fatal conclusion; these were naso-pharyngeal infection, *otitis media*, bronchopneumonia, stomatitis and urinary infection. In the later stages a patient might drift into a state of chronic enteritis (chronic ulcerative colitis) or food intolerance with marasmus; during this phase very careful feeding and general management were required to prevent infection and feeding upsets, which might cause death. In conclusion Dr. Crisp emphasized the importance of the following points: (i) a detailed early diagnosis, (ii) the treatment of toxæmia and dehydration by infusions *et cetera*, (iii) the use of the duodenal catheter, (iv) the dietetic management, particularly the apple diet and the avoidance of milk fats.

DR. DALY SMITH thought that it would be helpful if a standardized routine of treatment could be introduced into the hospital. He preferred his patients to be nursed at home unless they were dehydrated. He was interested in the question of diathesis.

DR. P. C. HOGAN asked about chronic cases in which convalescence was slow.

DR. HOBBY asked about the method of feeding breast-fed infants suffering from enteritis.

DR. H. J. DAVIS asked for a routine of treatment in mild cases.

DR. MYLES stressed the influence of the previous feeding and the state of nutrition.

Dr. Crisp, in reply to Dr. Daly Smith, said that a set scheme of treatment would be hard to lay down, because each case had to be judged by the child's previous feeding and state of nutrition, its age and its diathesis. An elastic scheme, based on sound general principles, could be drawn up. The slow, chronic case required regular routine examination to exclude parenteral infection. Sigmoidoscopic examination would often reveal chronic colitis with ulceration; "Tannalbin" was of great value in these cases, and the same applied to silver nitrate (one-sixth of a grain three times a day) given by mouth along with tincture of opium. Alkaline powders with bismuth, chalk, Dover's powder, and *Hydrargyrum cum Creta* were also of value. The diet should be low in fats and roughage, as had already been stated by Dr. Crisp, and should contain ample Calories in the form of food easily digested and absorbed from the upper portion of the bowel. In the case of breast-fed infants it was advisable to stop the breast milk temporarily, until the acute toxic stage had passed, and to resume it gradually, along with whey, buttermilk, or other food containing little fat. It had to be remembered that breast milk was very rich in fat and was badly tolerated in the presence of toxic enteritis. Naturally it was invaluable in the convalescent stages.

In reply to Dr. Davis, who had asked for a routine of treatment in mild cases, Dr. Crisp said that an initial purge should be given, perhaps with a bowel wash-out. For the first twenty-four hours the child should be starved, except for the fluids already mentioned (N/2 saline solution, glucose solution, apple juice or other fruit juice); perhaps the child might be given the apple diet. The medical attendant should see the child again in twenty-four hours. If the condition was satisfactory, an older child could be given cereals with water, clear broth *et cetera*, and a younger child could be given a low-fat milk such as buttermilk *et cetera*. Further treatment depended on the type of case and on the response to treatment. In conclusion, Dr. Crisp expressed his pleasure at being asked by the Goldfields members of the Western Australian Branch to address them.

The Effect of Dust on Miners' Lungs.

DR. K. R. MOORE showed a series of films drawn from the Commonwealth Health Laboratory, showing the development of industrial pulmonary fibrosis. He explained that the health laboratory had been established for fourteen years, and had records of the examinations

of miners entering the industry, as well as of those already employed. Over 30,000 men had been examined over that period. Dr. Moore had arrived at certain conclusions from observation of this series of cases. Pneumonokoniosis was described as being the presence of dust in the lung. Silicosis was that form of pneumonokoniosis due to the presence of silica. The diagnosis was made purely by X ray examination. A mottled film was necessary to establish the diagnosis. Workers might contract fibrosis with fine or coarse nodules. Workers in both coal dust and silica dust might develop nodular fibrosis of fine character, but workers in silica mainly suffered from coarse fibrosis. Dr. Moore said that stone-masons did not seem to get nodular fibrosis unless they were working in sandstone, which contained much free silica, so it appeared to him that the free silica content determined the nodular fibrosis. The fine type of fibrosis was rarely complicated by infection except late. It was relatively chronic, and incapacity was late; it occurred after thirty years or so, often in surface workers exposed to dust. The coarse type of fibrosis occurred relatively early (after about fifteen years or more). The patients suffered from early dyspnœa, sweats and blood-stained sputum, and died relatively early. Practically all specimens of sputum containing tubercle bacilli came from patients with the coarse type of silicosis, rarely from those with very old fine type, with late tuberculous involvement. This might be termed tuberculo-silicosis. The coarse type was rather to be called silico-tuberculosis. There was probably a pre-existing latent infective (perhaps tuberculous) process which determined the nature of the fibrosis.

Dr. Moore showed and discussed a series of radiographs demonstrating in various miners the development of silicosis from normality to definite tuberculosis. He stated and explained the rather arbitrary classification observed by him in his work. This was the following: (i) ante-primary silicosis (increased peribronchial thickening); (ii) primary silicosis (advanced to the formation of nodules); (iii) secondary or advanced silicosis (severe incapacity, which might be due to tuberculosis). Hilar shadows were not important.

DR. SMITH thanked Dr. Moore for his excellent paper, and asked for comments on the diathesis.

Dr. Moore said that he was still working on that question, and that he felt the catarrhal diathesis predisposed to the formation of silicosis. He indicated that in South Africa, workers with a phthinoid type of chest were rejected.

DR. P. C. HOGAN asked if an opinion could be given about the rate of development of fibrosis after a man left the mines.

Dr. Moore answered that there was not a great deal of evidence in his records to enable him to answer that question, but it was his opinion that the process in the ante-primary class would not advance after the men had left the industry.

DR. DONALD SMITH thanked Dr. Moore for his paper, and expressed the opinion that some original work had been performed in this field. He further congratulated him on the evenness of the films he had shown. He said that if there was no silica in the dust nothing else mattered very much, and he went on to discuss the influence of small particles of silica.

DR. JOHNSON asked why so many patients died of heart failure instead of toxæmia.

Dr. Moore said that patients whose sputum contained tubercle bacilli were usually dead in two years; if no tubercle bacilli were seen, they might die in the same way as patients with fibroid phthisis. Dr. Moore again pointed out the unique opportunity provided at the laboratory of working on patients, many of whom were new to the industry, working in pure silicosis dust, and who had a yearly reexamination. Coal miners could get nodular fibrosis, but without symptoms.

DR. SHANAHAN asked why the upper lobe of the right lung was so much more frequently involved than the left.

Dr. Moore would not venture an opinion.

The Royal Australasian College of Physicians.

SECOND ANNUAL MEETING.

THE second annual meeting of the Royal Australasian College of Physicians will be held in Melbourne on Friday and Saturday, April 5 and 6, 1940. The programme is as follows.

Thursday, April 4.

3 p.m.—Council meeting to be held in the Council Room of the Royal Australasian College of Surgeons.

Friday, April 5.

11 a.m.—Council meeting. The admission of new members will take place at this meeting.

12.45 p.m.—Luncheon at the Oriental Hotel.

2 p.m.—Annual general meeting to be held in the Lecture Hall of the Royal Australasian College of Surgeons.

2.45 p.m.—First scientific session in the Lecture Hall of the Royal Australasian College of Surgeons.

2.45 p.m.—Dr. F. M. Burnet: "The Problem of the Control of Pandemic Influenza".

3.30 p.m.—Lieutenant-Colonel E. Cooper: "The Technique of Micro-Radiography in the Routine Examination of Soldiers". The discussion will be opened by Dr. Reginald Webster, who will describe the bacteriological confirmation of the radiological diagnosis of incipient pulmonary tuberculosis.

4 p.m.—Afternoon tea.

4.30 p.m.—Professor R. D. Wright: "Secretion of the Small Intestine".

8.30 p.m.—Annual ceremony to be held in the Wilson Hall of the University of Melbourne. Academic and evening dress. At the conclusion of this function Fellows and Members and ladies accompanying them are invited by Dr. A. E. Rowden White to repair to the Union House, where light supper will be served.

Saturday, April 6.

10 a.m.—Second scientific session to be held in the Lecture Hall of the Royal Australasian College of Surgeons.

10 a.m.—Dr. T. E. Lowe: "Pathological Physiology of the Coronary Circulation".

10.30 a.m.—Dr. C. H. Kellaway and Dr. E. R. Trethewie: "The Liberation of Adenyl Compounds in Tissue Injury".

11 a.m.—Morning tea.

11.15 a.m.—Dr. A. J. Collins: "Syphilis of the Lung".

11.45 a.m.—Dr. E. B. Drevermann and Dr. Hilda J. Gardner: "The Treatment of the Schultz Syndrome (Granulocytopenia) by Marrow Extracts".

2 p.m.—Meeting of the combined councils of the Royal Australasian College of Physicians and the Royal Australasian College of Surgeons.

4 p.m.—Afternoon tea at "Road's End", Berwick. Fellows and Members and their wives are invited to this function by the President and Mrs. Sewell. Transport will be arranged for visitors from other States who desire to attend.

Post-Graduate Work.

WEEK-END COURSE AT ARMIDALE, NEW SOUTH WALES.

THE New South Wales Post-Graduate Committee in Medicine announces that, in conjunction with the Northern District Medical Association, it will hold a week-end course at Armidale on Saturday, April 13, and Sunday, April 14, 1940. The programme is as follows.

Saturday, April 13.

(At the Literary Institute, Armidale.)

2 to 2.45 p.m.—"Modern Views on the Cause and Treatment of the Toxæmias of Pregnancy", Dr. T. Dixon Hughes.
2.45 to 3.15 p.m.—Discussion.
3.15 to 4 p.m.—"Head Injuries", Dr. Douglas Miller.
4.30 to 5 p.m.—Discussion.
5 to 5.45 p.m.—"Ante Partum Hæmorrhage", Dr. T. Dixon Hughes.
5.45 to 6.15 p.m.—Discussion.
7.30 to 8 p.m.—"The Psychological Aspects of Hysteria", Dr. Duncan Howie (New England University College).
8 to 8.45 p.m.—"Intestinal Obstruction", Dr. Douglas Miller.
8.45 to 9.15 p.m.—Discussion.

Sunday, April 14.

9.30 to 10.15 a.m.—"The Management of Occipito-Posterior Positions", Dr. T. Dixon Hughes.
10.15 to 10.45 a.m.—Discussion.
11.15 a.m. to 12 noon.—"The Diagnosis and Treatment of Early Gall-Bladder Disease", Dr. Douglas Miller.
12 to 12.30 p.m.—Discussion.

The fee for the course will be one guinea. Those intending to be present at the course are requested to notify Dr. R. J. Jackson, Honorary Secretary of the Northern District Medical Association, Armidale, as soon as possible.

Correspondence.

PINK DISEASE TREATED BY VITAMIN *B*.

Sir: The following two cases are a continuation of a series of four published in this journal on November 18, 1939, under the caption of "Pink Disease Treated by Vitamin B_1".

Both these children improved under wheat germ therapy in the same manner as those whose histories were previously published. The dose, however, was greater than in the previous series. Measured in terms of vitamin B_1 content, the daily dose was 1,200 to 1,500 units. I am, however, inclined to the opinion that some other factor beside B_1 is important in this therapy.

Case V occurred in my own series. The clinical notes for Case VI were kindly supplied to me by Dr. C. S. Fitzpatrick, of Hamilton, Victoria.

Case V.—T.G.F., a male child, aged eleven months, was suffering from typical pink disease when first seen on September 15, 1939. There was a six weeks' history of gradually increasing illness, initiated, according to the history given by the mother, by a severe skin eruption resembling measles, which lasted a few days only and was replaced by a much finer rash on the trunk and limbs. The child had become disinterested, irritable and sleepless, and had developed a pinkness of its hands and feet. Its weight had decreased from 22 pounds 6 ounces at ten months to 18 pounds 4 ounces at eleven months. The child had been breast fed up to eight months.

On examination it was drowsy and disinterested, but irritable, and cried in a peevish whimper when examined. Its limbs were tender and hypotonic. Sweating, a sudaminal rash and very pink hands and feet were prominent signs. Photophobia was present. The left *membrana tympani* was slightly reddened, but not bulging.

On September 15, 1939, one ounce of "Bemax" was ordered three times a day (1,200 units of vitamin B_1 *plus* other factors) and five drops of "Halivol" twice daily. No change was made in the dietary and no other treatment was ordered.

Four days later the child was slightly worse; on the fifth day his condition was definitely improved. The improvement continued, and ten days after the commencement of "Bemax" therapy the child was sleeping peacefully at night, taking an interest in its toys, and its hands and feet had assumed a normal colour. Sweating and photophobia had disappeared, but the sudaminal rash persisted.

Fourteen days after commencement of treatment the child looked well and its weight had increased eight ounces. It refused to take "Bemax", and "Cenovis Yeast" (nutritive) was substituted in doses of one heaped teaspoonful night and morning (about 1,500 units of *B* and 1,000 units of B_2, together with other factors). The general health rapidly improved, the weight increasing to 19 pounds 5 ounces by October 3, 1939. The sudaminal rash disappeared shortly after yeast therapy was commenced, and its limbs regained their tone and strength. By October 23, 1939, the baby weighed 20 pounds 1 ounce and slept from 7 p.m. to 8 a.m. and from 9 a.m. to midday.

Case VI (Dr. Fitzpatrick's case).—B.M., a female child, aged fifteen months, the third in a family in comfortable circumstances, was first seen on October 4, 1939. It was breast fed for twelve months and then a liberal diet was begun for her age. Ten weeks before, it had developed "measles", which was diagnosed by the mother; the child was not seen by a medical man. It is very doubtful whether the condition was not rubella rather than morbilli, or it may have been the sudaminal rash of pink disease. Ever since she has lost weight, strength and cheerfulness, has become flabby and had a loose cough, anorexia and constipation.

On examination her temperature is 98·8° F., her pulse rate is 110 in the minute, and she is pale and thin, with a flushed patch on her cheeks, a perspiring head, photophobia and poor muscle tone. She is somewhat puffy. Her weight is 19 pounds 12 ounces (it had been up to 28 pounds). She is sensitive to egg albumen.

On October 13, 1939, one ounce of "Bemax" was ordered to be fed to her three times a day. In five days the mother reported a great change for the better, and then the child developed measles, together with two other children in the family. This rather put her back; but by October 27, 1939, the child was totally changed; misery had changed to an active cheerfulness; she was smiling and crowing and was sleeping the whole night; she had no sweating; the hands and feet were normal; the weight was 20½ pounds. There was some slight wheezing, but the chest signs were almost clear.

Yours, etc.,

G. Forsyth.

Lister House,
Horsham,
Victoria.

BLUE-DOMED CYSTS OF THE BREAST AND CARCINOMA.

Sir: Now that women are more often having any lump they may find in the breast seen to with but little delay, a greater responsibility has been thrown on all of us to make a correct and prompt diagnosis.

Careful consideration of all the clinical evidence will allow one to reach an accurate conclusion in the great majority of cases, and the small residuum can usually be sorted out while the patient is still on the table by examination of a frozen section and by the macroscopic appearance of the mass after it has been cut into. In a very small minority it is necessary to wait for a report on a paraffin section.

Bloodgood is a name to conjure with in breast pathology, and he has called one type of breast cysts "blue-domed", because, when carefully exposed, they have a distinct blue look, which disappears as soon as the cyst is cut into. Others have a white dome, and Bloodgood has called these "cysts of the galactocele type". The former are quite common.

Backed by his authority one has sheltered under such statements as the following: "This and other observations

have led me to conclude that there is no relationship between single and multiple blue-domed cysts and the cysts of the galactocele type and carcinoma. If exploratory incision reveals a cyst of this character there is apparently no necessity for removing one or both breasts." Again in the same paper the following occurs: "Now my evidence convinces me that the blue-domed cyst is so characteristic of benignity that one may excise it with a zone of breast tissue unopened" *et cetera*.

I must confess that the dogmatism limited my faith somewhat, for when it comes to sweeping statements in surgery I am reminded of the wise saying that there is only one "never" in bridge, and that is: "Never fail to make the ace of trumps." However, for years one acted on the idea that where there were cysts of this nature there was probably no carcinoma; but in the last few months I have had two rude shocks to this simple faith.

In the first case I removed a doubtful mass from the breast containing what looked a typical blue-domed cyst. On opening the cyst it was found to contain not fluid but mucoid, somewhat granular material, in the identification of which a frozen section did not help. On the macroscopic appearance it was decided to do a radical operation, and the wisdom of this course was proved when, on examining a paraffin section, Dr. Davies pronounced it to be a mucoid carcinoma. The second case also had a typical blue-domed cyst, but on cutting into it the tissue was clearly carcinomatous and a radical operation was done immediately. Dr. Davies reports this as a carcinoma apparently originating in a duct papilloma.

Safety apparently lies in opening all blue-domed cysts found at exploratory operation, and also in having the help of a pathologist in the theatre in deciding the probable nature of doubtful lumps in the breast.

Yours, etc.,

MAYNARD FURBER.

143, Macquarie Street,
Sydney.
(Undated.)

TICK BITE AND ALLERGY.

SIR: I have read in the "Current Comment" (March 2), Robert Benson's views, that the antigens of insects causing violent reactions are probably essentially different from those causing the ordinary mild reactions of the same insect; and that the violent reactions are due to some general body substance in the insect and not to the venom that causes the milder reactions. Let us see how this idea does not work out when we consider the events following tick bite.

The dog-tick (*Ixodes holocyclus*) so common about Sydney, is of course not an insect, but is grouped with spiders and scorpions under the Arachnida. A tick does not "bite", she has no head; but she, like the mosquito, injects her venom into her host and then the trouble starts.

Since I wrote a monograph on the anatomy and poison of the dog-tick (1928) I have tried to explain the signs and symptoms following tick-bite, which I now consider due to the allergens injected. I may say that I follow A. H. Rowe, who in the last edition of his great work on clinical allergy declares that "allergy in man and animals is fundamentally the same" and "allergy and anaphylaxis are fundamentally identical".

The first diagnostic symptom that a man exhibits after the tick bite may show in the form of urticaria and angioneurotic œdema, and I have notes of a case where symptoms were developed within a few hours. The symptoms in a dog show up, if only one tick bites, within forty-eight hours; and the first definite symptom is that the dog refuses to take a drink, even in the hottest weather.

After watching one of my own dogs that had been bitten by two ticks I came to the conclusion that the reason why a dog and a child refuse to drink was that the vagus was affected. Abundant observations have confirmed that, and I now declare that a child or a dog bitten by the tick *Ixodes* suffers because of the occurrence of vagotonia, which leads to spasm or paresis of the muscles of the gullet, while in children there is at the same time developed sympathicotonia—both conditions being the result of the allergens, or as some prefer, the antigens in the salivary secretion injected by the tick.

The vagotonia is shown in two ways; a dog will not drink, for he knows after his first attempt that the fluid goes the wrong way; and this is why so many dogs die of pneumonia when the kind-hearted owner pours fluid into their throats, for it enters not the gullet, but the trachea, the muscles of both being probably affected.

The vagotonia is exhibited in another way. I took my dog and placed her near another dog and told the yardman to disguise himself and come toward the dogs; the unaffected dog barked with fury; the dog with vagotonia tried to bark, but no sound came; both recurrent laryngeal branches of the vagus on both sides had paresis from the allergens in the tick venom. The sympathicotonia is well shown in some cases of children bitten by ticks, in which it is recorded that they would not take even a sip of water and that their bowels worked ten times a day. Monrad-Krohn enumerates mucous colitis among the symptoms of parasympathetic irritability. I suggest that it is an irritation of the sympathetic.

These symptoms are not peculiar to children bitten by our dog-tick, for in Canada, where two different species of tick (*Dermacentor* and *Ornithodorus*) bite the children, the same events happen. Here is an extract I made from one Canadian report: "Thorax and larynx were involved; her breathing was laboured, and she was unable to swallow liquid; phonation was impossible, and she could make only low guttural sounds." All these symptoms were due to vagotonia.

Joseph Bancroft, of Queensland, first described "tick blindness". This has always been regarded with suspicion; but why should it be? Albert Rowe assures us that "ocular disturbances including transient blindness may follow food allergy". Why should blindness not follow the injection of an allergen by a tick?

We know that the bites of household ticks cause asthma, the king of the allergies; that the bites of bed-bugs also cause asthma, while allergy manifestations may be caused by the caddis fly, by ascaris, by echinococcus and oxyuris and many other parasites; and so it is not wonderful that the dog-tick when she bites produces urticaria, angioneurotic œdema and vagotonia.

Benson's idea that the violent reactions are due to some general body substances in the insects and not to the venom which causes the milder reactions does not appeal to me when one sees what the following evidence shows: (*a*) Three hundred seed ticks have bitten an adult at the same time and caused marked urticaria, nothing else. (*b*) A nymph tick (the next stage) bites a dog, and Clunies Ross has observed slight paresis afterwards, as well as urticaria. (*c*) If one mature tick bites a dog he may become paralysed and may recover, but often dies. (*d*) If three female ticks bite a dog at once, he dies on the third day. (*e*) A tick bites a child, the child may recover or die. (*f*) A tick bites a man, and while he may exhibit signs of urticaria and angioneurotic œdema, he never dies.

The above evidence points to the salivary venom as being the cause in the mild and severe symptoms, the strength of the allergen injected, the amount, and the size of the animal attacked being all important factors in the final result. Lastly, the allergen injected into a vein works more quickly than if injected subcutaneously.

I now bring forward evidence which will show beyond all doubt that it is the salivary secretion that is responsible for the violent reactions in the tick.

If one dog-tick is placed on a guinea-pig and becomes attached, the guinea-pig shows no symptoms until the fourth day. On that day and the next the tick takes in much blood and is as full as she can be on the fifth. If the salivary glands of the tick are observed it will be seen that they increase every day in size. On the fifth day the tick gives the guinea-pig more venom and the pig changes rapidly and dies on the night of the fifth day.

It almost looks as if the first injections given sensitized the guinea-pig, and the injections on the fifth day killed it by anaphylactic shock.

The blood taken in by the tick is the pabulum, which goes to form the two thousand eggs; but first of all it supplies the tick with a new stock of venom.

Yours, etc.,
W. J. STEWART McKAY.

Lismore,
New South Wales.
(Undated.)

Naval, Military and Air Force.

APPOINTMENTS.

THE undermentioned appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Number 49, of March 14, 1940.

PERMANENT NAVAL FORCES OF THE COMMONWEALTH (SEA-GOING FORCES).

Surgeon Lieutenant (for short service) Godfrey Joseph Kelleher Lane is appointed Surgeon Lieutenant on the Permanent List, with seniority in rank of 30th September, 1934, dated 30th March, 1940.

AUSTRALIAN MILITARY FORCES.

EASTERN COMMAND.

Second Military District.

Australian Army Medical Corps.

To be Captain (provisionally)—Ewan Lawrie Corlette, 12th November, 1939. Captain W. M. Ada is appointed from the Reserve of Officers (A.A.M.C.), 12th November, 1939.

To be Captain (provisionally) supernumerary to establishment pending absorption—Howard Hamilton Webber, 19th September, 1939.

SOUTHERN COMMAND.

Third Military District.

Australian Army Medical Corps.

To be Major (temporarily)—Captain L. Smith, 22nd January, 1940. *To be Captains (provisionally)*—Norman Franklyn Freemantle and Alan Godfrey Gordon Carter, 31st December, 1939, and 10th February, 1940, respectively. Honorary Captains L. G. Travers and R. B. Perrins are appointed from the Reserve of Officers (A.A.M.C.) and to be Captains (provisionally), 12th November, 1939, and 11th February, 1940, respectively.

Australian Army Medical Corps Reserve.

To be Honorary Captains—Edgar Livingstone Davey, John Daniell Begg, Sydney Meredith Hall, Bryan Joseph Mahon, Edmund John Pickford and Henry Elford Rowe, 21st February, 1940.

WESTERN COMMAND.

Fifth Military District.

Australian Army Medical Corps.

To be Majors (temporarily)—Captains H. H. Stewart and R. G. Williams, 15th February, 1940.

ROYAL AUSTRALIAN AIR FORCE.

Citizen Air Force.

Medical Branch.

Ian Lindemann Miller, M.B., B.S., is granted a commission on probation with the rank of Flight Lieutenant with effect from 26th February, 1940.

The following are granted commissions on probation with the rank of Flight Lieutenant with effect from 4th March, 1940: Rollo Greenlees, M.B., and Albert Norman Poulton, L.D.S., B.D.S. John Dennis Gribble Dunn is granted a commission on probation with the rank of Flying Officer with effect from 4th March, 1940; Sydney Bayly Forgan, M.B., B.S., is granted a commission on probation with the rank of Flight Lieutenant with effect from 13th March, 1940; Lionel Pelham Sapsford, M.B., B.S., is granted a commission on probation with the rank of Flight Lieutenant with effect from 21st March, 1940.

Reserve.

Medical Branch.

Noel Millar Cuthbert, M.B., Ch.M., D.L.O. (London), F.R.A.C.S., is granted a commission on probation with the rank of Flight Lieutenant and the honorary rank of Squadron Leader with effect from 26th February, 1940. Howard John Edelman is granted a commission on probation with the rank of Flight Lieutenant with effect from 26th February, 1940. Kenneth Edward Rex, M.B., B.S., is granted a commission on 1st March, 1940.—(Ex. Min. No. 14.—Approved 12th March, 1940.)

Proceedings of the Australian Medical Boards.

NEW SOUTH WALES.

THE undermentioned have been registered, pursuant to the provisions of the *Medical Practitioners Act*, 1938-1939, of New South Wales, as duly qualified medical practitioners:

McVeigh, James Samuel, M.B., Ch.B., 1938 (Univ. New Zealand), Royal Prince Alfred Hospital, Camperdown.

Wilson, Thomas Edward, M.B., B.S., 1936 (Univ. Melbourne), Prince Henry Hospital, Little Bay.

Young, Geoffrey Lawrence, L.M.S.S.A. (London), 1938, Katoomba.

TASMANIA.

THE undermentioned have been registered, pursuant to the provisions of the *Medical Act*, 1918, of Tasmania, as duly qualified medical practitioners:

Lauricella, Salvatore Gaetano, M.B., B.S., 1937 (Univ. Melbourne), Launceston.

White, Stanley Boyd McKellar, M.B., B.S., 1939 (Univ. Sydney), Royal Hobart Hospital, Hobart.

Isles, Jack Dennis, M.B., B.S., 1939 (Univ. Melbourne), Royal Hobart Hospital, Hobart.

Merry, John William Hugh, M.B., B.S., 1939 (Univ. Melbourne), Royal Hobart Hospital, Hobart.

Mercer, David Lloyd, M.B., B.S., 1939 (Univ. Melbourne), Launceston.

Mulcahy, Francis John Xavier, M.B., B.S., 1935 (Univ. Melbourne), Launceston.

Dorney, Paul Laurence, M.B., B.S., 1937 (Univ. Melbourne), Hobart.

Walker, Morris Arthur, L.R.C.S. (Edinburgh), 1932, L.R.C.P. (Edinburgh), 1932, L.R.F.P.S. (Glasgow), 1932, Hamilton.

Obituary.

VINCENT JULIAN BRYANT.

WE regret to announce the death of Dr. Vincent Julian Bryant, which occurred on March 21, 1940, at Sydney, New South Wales.

Corrigendum.

AN error occurred in the issue of March 16, 1940, on page 395, under the heading of "Naval, Military and Air Force: Appointments". The announcement in connexion with Command Headquarters Staff of the Fourth Military District should read as follows:

Lieutenant-Colonel F. H. Beare, E.D., Australian Army Medical Corps, is appointed Deputy Director of Medical Services and is granted the rank of Temporary Colonel, 27th November, 1939, *vice* Colonel S. R. Burston, C.B.E., D.S.O., V.D., seconded to the 2nd A.I.F.

Books Received.

STEDMAN'S PRACTICAL MEDICAL DICTIONARY, by T. L. Stedman, A.M., M.D., and S. T. Garber, B.S., M.D.; Fourteenth, revised, Edition; 1939. London: Baillière, Tindall and Cox. Super royal 8vo, pp. 1316, with 23 plates and numerous illustrations. Price: 37s. 6d. net.

Nominations and Elections.

THE undermentioned has applied for election as a member of the Victorian Branch of the British Medical Association:

Harvey, Rupert F., L.R.C.P. and S. (Edinburgh), L.R.F.P.S. (Glasgow), 1930, 6, Whernside Avenue, Toorak, S.E.2.

The undermentioned has applied for election as a member of the New South Wales Branch of the British Medical Association:

Buckley, Joseph Gregory, M.B., B.S., 1936 (Univ. Sydney), 112, Willison Road, Carlton.

Diary for the Month.

APR. 2.—New South Wales Branch, B.M.A.: Council.
APR. 3.—Victorian Branch, B.M.A.: Branch.
APR. 3.—Western Australian Branch, B.M.A.: Council.
APR. 4.—South Australian Branch, B.M.A.: Council.
APR. 5.—Queensland Branch, B.M.A: Branch.
APR. 9.—New South Wales Branch, B.M.A.: Executive and Finance Committee; Organization and Science Committee.
APR. 9.—Tasmanian Branch, B.M.A.: Branch.
APR. 12.—Queensland Branch, B.M.A.: Council.
APR. 16.—New South Wales Branch, B.M.A.: Ethics Committee.
APR. 17.—Western Australian Branch, B.M.A.: Branch.
APR. 18.—New South Wales Branch, B.M.A.: Clinical Meeting.
APR. 23.—New South Wales Branch, B.M.A.: Medical Politics Committee.
APR. 24.—New South Wales Branch, B.M.A.: Branch.
APR. 24.—Victorian Branch, B.M.A.: Council.
APR. 25.—South Australian Branch, B.M.A.: Branch.
APR. 26.—Queensland Branch, B.M.A.: Council.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser", pages xiii-xv.

AYR HOSPITALS BOARD, AYR, QUEENSLAND: Junior Medical Officer.

MOUNT BISCHOFF PROVIDENT HOSPITAL, WARATAH, TASMANIA: Medical Officer.

PRINCE OF WALES REPATRIATION GENERAL HOSPITAL, RANDWICK, NEW SOUTH WALES: Consultant Anæsthetist.

STATE GOVERNMENT OF WESTERN AUSTRALIA: Inspector-General of the Insane.

TARA DISTRICT HOSPITAL, TARA, QUEENSLAND: Medical Officer.

THE BRISBANE AND SOUTH COAST HOSPITALS BOARD, QUEENSLAND: Junior Surgeon, Assistant Surgeon.

THE RACHEL FORSTER HOSPITAL FOR WOMEN AND CHILDREN, SYDNEY, NEW SOUTH WALES: Medical Officers.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment referred to in the following table without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCHES.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	Associated Medical Services Limited. All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Federated Mutual Medical Benefit Society. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17.	Brisbane Associate Friendly Societies' Medical Institute. Proserpine District Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.
SOUTH AUSTRALIAN: Secretary, 178, North Terrace, Adelaide.	All Lodge appointments in South Australia. All Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 205, Saint George's Terrace, Perth.	Wiluna Hospital. All Contract Practice Appointments in Western Australia.

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